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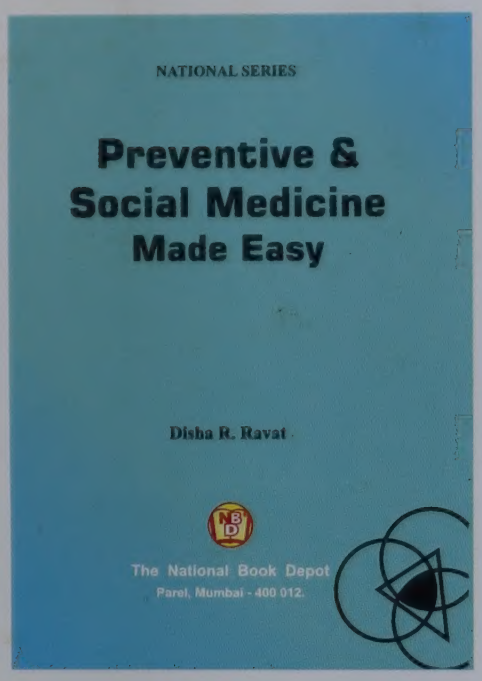

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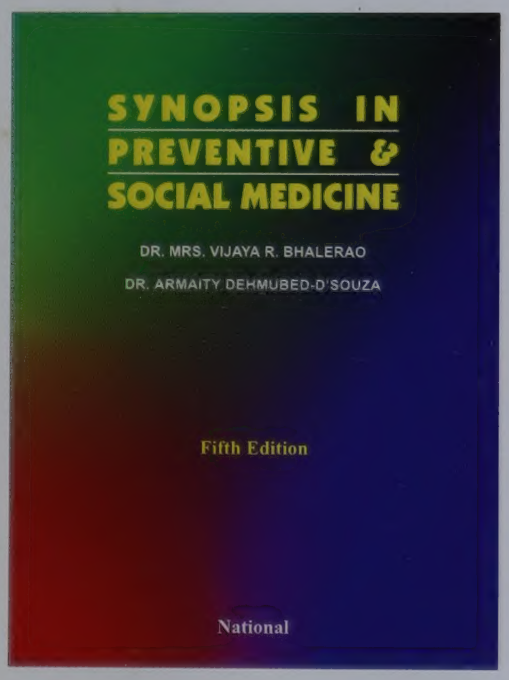

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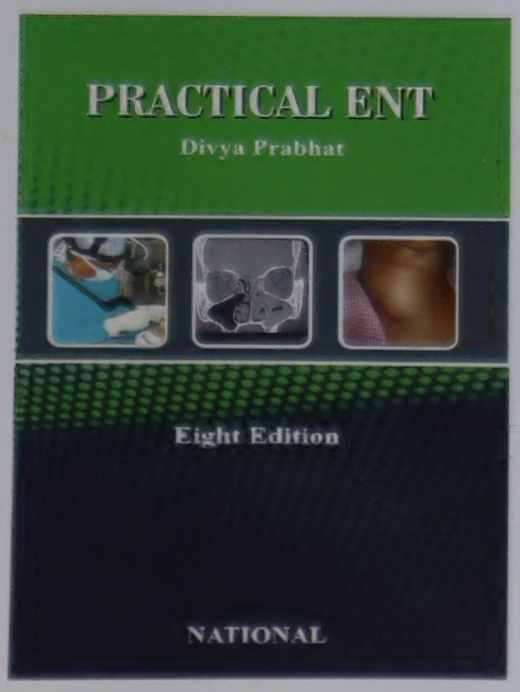

PRACTICAL
PREVENTIVE
&
SOCIAL MEDICINE

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our
Parents

Shri. Srinivas Shastri
And
Smt. Kamal S. Shastri

~~~~~~~~~~~~~~~~~~~~

Dr. Chaturbhai Patel
And
Smt. Kamlaben Patel

~~~~~~~~~~~~~~~~~~~~

Shri. Mohanlal Rathod
And
Smt. Jayaben Rathod

FOREWORD

For a student appearing for his examination, Preventive and social Medicine is considered to be an unwieldy subject. Epidemilogical details of diseases, various national programmes and bio-statistics are formidable subject to understand and recollect at the time of examinations. This book aims to help the student in appearing for examinations with confedence by preparing him thoroghly for the practicals. The contents are well planned and will guide the students step by step through the various parts of the practical examinations. The book is easy to read and follow and the addition of tables and flow charts will help the students to grasp different concepts easily. I am certain that the medical students will find this book of great help.

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PREFACE

our experience as teachers at the Seth G.S. Medical College and as an examiner to several universities, have found that medical students are initially interested in the subject, but fail to get the necessary formation from their teachers/text books, and hence turn away from the subject. Ultimately, the dents merely want to clear the obstacle of PSM, so that they can move ahead in their career. It shall ways remain our endeavor to make the subject as simple and interesting as possible.

e wish to thank our students for making our first book "Practical Book of Preventive and Social Medicine" great success. The book has been in great demand since it's publication. This new book titled simply as CLINICAL BOOK OF PREVENTIVE & SOCIAL MEDICINE" is a sequel to the earlier book and contains l essential information needed for PSM Practicals exams.

e thank our Dean Dr. R.G. Shirahatti for writing a foreword to this book.

pecial thanks are due to our friends whose feedback on the previous book has been of great help to us:

Dr. P.S.N. Reddy	Dr. N. Pai	Dr. M.R. Jape	Dr. P. Subramanian
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			Mr. Bharat Vyas

Very special thanks to all the members of Shastri, Patel and Rathod families for bearing with us most patiently and letting us work in peace at the most odd hours.

Dr. Surendra Shastri

Dr. Kirti Patel

Dr. Nitin Rathod

INTRODUCTION

reventive and Social Medicine has made its entry in the Medical curriculum in the seventies. reviously it was a part paper in General Medicine and later got recognition as a separate subject. Till the id-eighties it was a subject at the IInd M.B.B.S and was latershifted to IIIrd M.B.B.S. As per the Medical ouncil of India directives, PSM teaching should begin in the 1st M.B.B.S., continued through the IInd .B.B.S and the examination is to be held in the IIIrd M.B.B.S.

t present most of the Indian Universities follow the MCI pattern of curriculum. The examination held at IIrd M.B. B.S usually consists of theory and practicals carrying equal marks (usually 100 each). Some niversities assign 20% of marks in theory and practicals to internal assessment. Theory papers are sually partly essay type and partly objective. Practicals are always extensive and have a definite pattern. Practicals consist of

(a) Case examination, presentation and viva.
(b) Statistics.
(c) Spots.
(d) General viva.

Each of these carry specific marks, and a student has to master all of them.

It has been our experience that the student is many a times unsure about what is expected out of him in a practical examination, resulting in confused responses to most easy and simple questions. Another problem which is specific to the subject PSM is that a medical student gets very little scope, to practice what he learns in the subject, in his later medical career. This forms a strong disincentive, for the student to devote much time towards this subject. Towards the end the student just memorizes the important points in an attempt to clear the IIIrd M.B.B.S examination. The student never realises the applied importance of the subject.

There has been since a longtime, a real need of a hook on the PSM practicals. This book is expected to fulfill this longstanding requirement.

CONTENTS

I. CASE EXAMINATION AND VIVA

The case examination in PSM is different from that in other medical subjects. The PSM teacher is more interested in the epidemiological aspects of the case; i.c. the important, Agent, Host and Environmental factors relevant to the disease. The student should learn to highlight these, rather than routinely narrate all possible environmental and social factors which may have absolutely no connection with the disease under discussion. Socio-economic aspects and living conditions including the housing, ventilation, over-crowding, nutritional status, water and food hygiene may be very important in some diseases while they may not have any relevance with many others.

The student should first think of the mode of tranmission of the disease which he has diagnosed, and then highlight only those epidemiological aspects which could be responsible for the disease.

In the following pages we have discussed the important epidemiological aspects of the diseases which are commonly kept for case examination in PSM. The student should try to identify these in his case.

The student will then have to discuss in his viva, statistics related to the disease as well as the usual clinical features, complications, investigations to be carried out and the management of the case.

Finally he has to discuss the prevention and control of the disease in the community at large. All these aspects are discussed in details against the relevant diseases. Other than these topics the student is expected to know the rehabilitation centres and institutions of spccial interest where he can refer the case if required. These institutions are usually ones where the students are taken for visits during, their PSM training.

Example

(I) All India Institute of Physical Medicine and Rchabilitation at Haji Ali for the physically handicapped patient.

(II) Vakil's school at Sewri and Home for mentally retarded at Mankhurd, for those with a mental handicap.

(III) Ali Yavar Jung Institute for auditory and speech handicap.

(IV) Blind school at Worli and Dadar for the visually handicapped.

(V) Acworth leprosy hospital for the treatment and rehabilitation of leprosy patients.

(VI) Kasturba Hospital, where infectious diseases patients are referred.

(VII) Alcoholics anonymous centres,Narcotics anonymous centres and centres for the rehabilitation of drug addicts.

(VIII) Agencies running Orphanages and offering adoption services.

(IX) Agencies offering Medical Termination of Pregnancy services.

An occassional teacher also likes to ask about.

(A) Socioeconomic status scale: Kuppuswamy's classification considers the
  (i) per capita income.
  (ii) occupation
  (iii) educational status

While Prasad's clasificaiion considers only the per capita income.

(B) Overcrowding: May be defined as less than
  i) Minimum 75 to 100 sq. ft. per person
  ii) 1 room for 2 persons.
    2 rooms ror 3persons.
    3 rooms for 5persons etc.
  iii) Also if 2 persons above 9 yr. of age of opposite sex, who are not husband and wife are obliged to sleep in the same room.

(C) **Poverty Line:** The World Bank's definition of the poverty line, for under developed countries, like India, is US$ 1 per day per person or US$365 per year. As per this definition, more than 75% of all Indians are, probably, below the poverty line. As per the Government of India, poverty line for urban areas is Rs.296 per month and for rural areas Rs.276 per month, i.e. people in India who earn less than Rs.10 per day (Rs.3650 per year or US$ 75 per year). As per the government, this amount is enough to buy food equivalent to 2200 calories per day, apparently enough food to prevent death from starvation (sic). On what basis have our planners decided this definition of "Poverty Line", and what about a persons minimum needs in terms of education, housing, clothing and health services. Even by this definition the number of people living below the poverty line are around 300 million (30 Crores) or 30% of the population.

* **Remember that most of the discussion will be on the epidemiology, prevention and control.**

1. Coronary Heart disease

A. Statistics: A WHO project termed MONICA (Mullinalional moniloring of trends and determinants in cardio-vascular disease) involving 26 countries has revealed the following.

Annual incidence around
1.5 to 7.5 % (Males) and
0.5 to 1.5 % (Females) in the West.

Incidence figures are not available for India. However, a few crossectional surveys with ECG revealed that the prevallence of CHD was
6.5% urban males.
4.5% urban females.
2.5% rural males.
1.5% rural females.

B. Epidelniology :

I. **Agent:** No single agent can be pinpointed as the causive agent for coronary heart disease. The disease is caused by the interaction of a variety of factors. Hence it is usual to consider the disease causation for CHD in terms of certain risk factors (relaled to the host) rather than a single agent.

II. **Host:** Look for the following risk factors in the history.
a. Age : 40 plus.
b. Sex : predominantly male
Post menopausal females.
c. Personality : Type 'A'
d. Family History of CHD.
The above factors are considered non-modifiable since, none has any control over them.
The factors which can be controlled are called modifiable risk factors. Look for the following modifiable risk factors in the patients history.
i) Stressful living style.
ii) Heavy smoking.
iii) Hypertension.
iv) Obesity & sedentary life style.
v) Diabetes.
vi) Raised serum cholestrol levels particularly those of low density Lipoprotein. Cholesterol is a confirmed atherogenic factor, but cannot hc elicited in the history, unless the patient has already under gone these investigations previously.
vii) Oral Contraceptive Pills.
viii) High serum homocystine levels

C. Clinical features :

1. **Angina:** Chest pain usually occurs in lower sternum or over precordium. It may radiate anywhere from umbilicus to lower jaw. It is a vice-like, constricting or choaking sensation or just heaviness of chest. It may last from 1 to 5 minutes and gets immediately relieved with sublingual administration of nitrate.

2. **Unstable Angina:** This condition includes the following.
a. Patient with recent onset angina.
b. Angina occurring at rest
c. Chronic stable angina with recent increase in intensity, duration and frequency of pain.
d. Post-myocardial anginal pain occuring within 6 months of attack.

3. **Acute myocardial Infarction :**
a. Chest Pain: Retro-sternal which may radiate to left arm.
b. Nausea, vomiting.
c. Profuse sweating,
d. Shock (Depending upon degree of Infarction.)

Complications of Myocardial infarction:

Early:
a. Arrythmias
b. Cardiac failure
c. Embolisation
d. Cardiogenic Shock
e. Cardiac rupture
i) Ventricular Septal Defect
ii) Rupture through free wall causing cardiac tamponade.
f. Papillary muscle dysfunction.

Late :
a. Ventricular aneurysm.
b. Dressler's syndrome
c. Shoulder hand syndrome.

D. Investigations:

I. Angina:
a. Electrocardiogram is usually normal but may show ischaemia
b. Computerised Stress Test
c. Radionuclide studies
Thallium-201 radioactive substance is used for myocardial imaging.
d. Echocardiography.
e. Coronary Angiography.

2. Unstable Angina and Mycardial Infarction:

a. Leukocytosis with high ESR
b. Serum enzymes
   i) Raised MB fraction of CPK
   ii) Raised SGOT (serum Glutamic oxalate transferase)
   iii) Raised LDH (Lactic dehydrogenase)
c. Electrocardiogram
d. Coronary Angiography.

E. Medical treatment

I. For Angina

a. Control of risk factors like Hypertension, Diabetes etc.
b. Bed rest & sedativcs
c. Nitrates - Isosorhide dinitrate - Nitroglycerine ointment 2%
d. β - blockers
e. Calcium channel blockers.

II. For Unstable Angina and Myocardial infarction.

a. Relief of pain:
   i) Complete bed rest
   ii) Nitrates - sublingually
   iii) Analgesics
   iv) Nitrate - Intravenous
   v) Heparin
   vi) Thromholytic therapy
b. Supportive treatment
   i) Prevention of deep vein thrombosis
   ii) Proper laxatives
   iii) Liquid diet initially during attack.
   iv) Sedative to allay the anxiety.
   v) Treatment of complications like failure, arrythmia etc.
c. Percutancous Transluminal Coronary Angioplasty (PTCA)

F. Surgical treatment

Coronary arterial Bypass.

G. Prevention and Control

I. Primary Prevention

1. Hcalth Promotion:
a. *Health education* :
   i) Creating awareness about the disease.
   ii) Risk factors to be avoided. Preventing the emergence of risk factors and life style changes is also called 'Primordial Prevention'.

a) Regular excrcises and Yogasanas are useful.
b) Keeping hypertension and diabetes under constant check and under control.
c) Avoid undue stress.
d) Keep smoking and alcohol within strict control or totally avoid them. The Framinghanm study, North Karelia (Finland) project, and the Multiple Risk Factor Intervention Trial (USA) are some of the famous risk factor modification, interventional studies.

   iii) Training of volunteers and public functionaries in the techniques of External Cardiac Massage and artificial respiration can save many lives.

b. *Legislation:*
   i) Banning the sale of cigarettes & alcohol
   ii) Making compulsory, the printing of the statutory warning that smoking and drinking alcohol is injurious to health.
   iii) Banning the advertisment of cigarettes and alcohol.

2. Specific protection:
   a. The use of garlic extract & certain gums e.g. yograj guggul has been shown to reduce the cholesterol level in the blood.
   b. The use of oils containing polyunsaturated fatty acids. e.g.
      Groundnut oil.
      Sunflower oil. Safflower oil.
   c. Limiting the fat intake so as not to exceed 30% of the daily calorie requirement.

II. Secondary Prevention

1. Early diagnosis:
   a. High risk screening: screening of population groups who are known have many of the risk factors e.g. office executives.
   b. Routine periodic investigations of persons with a family history and with other known risk factors.
   c. Cardiac stress test: Bicycle, Treadmill and Masters step test to unmask the early cases showing normal routine ECG.
2. Prompt and Effective treatment: Treatment on the lines discussed above with diet management & lifestyle changes. Regular follow ups and routine periodic checkups.

III. Tertiary prevention

1. Disability limitation:
   a. Present techniques available are Balloon Angioplasty and Cardiac Bypass Surgery.
   b. Laser and Ultrasonic destruction of clots would be techniques available in the future.
2. Rehabilitation:
   a. Physical rehabilitation by physiotherapy would be required in most cases.
   b. Occupational rehabilitation in some cases.
   c. Psychological rehabilitation would be required in about 60 -70% of the cases.

2. Hypertension

A. Statistics:

It has world wide prevalence. A 'rule of halves' or 50% has been worked out for this disease which says that

50% of the patients were aware of their state,
50% of those aware were taking treatment.
50% of those being treated were being treated properly.

The prevalence is estimated at 15-20% of the adult population in developed countries. Two studies in India suggested a prevalence of 6-7% in adult males and 3-4% in adult females.

Although morbidity due to the disease has increased tremendously in both the developing and developed countries, the mortality has significantly decreased in the developed countries due to better awareness, detection techniques and treatment facilities.

B. Epidemiology:

In this disease, no single agent can be identified. The epidemiology is discussed in terms of certain risk factors related to the host.

Host

1. **Risk factors** (non modifiable)
   a. Age: Increases with age in both sexes.
   b. Genetic factors
2. **Risk factors** (modifiable)
   a. Obesity
   b. High salt intake
   c. Diet with a high content of saturated fats
   d. Alcohol
   e. Smoking
   f. Lack of physical activity
   g. Oral contraceptives, corticosteroids etc
   h. Mental stress.

C. Clinical features:

1. Asymptomatic, detected on routine check-up.
2. Headache in the occipital region occuring in the morning.
3. Dizziness.
4. Palpitation.
5. Easy fatigability.
6. Epistaxis.
7. Angina pectoris.

D. Investigations:

1. Urine Examination for renal disease.
2. Serum Electrolyte: Hypokalemia may be present in Aldosteronism.
3. Complete blood count.
4. FBS/PPBS: Increased blood sugar levels are found in Cushings and Pheochromocytoma.
5. Blood Urea Nitrogen and Creatinine; It increases in renal failure.
6. ECG - It reveals left ventricular strain.
7. X-ray chest: It may be useful in diagnosing Aortic aneurysm or Rib notching.
8. Special investigations include
   a. Intra-venous Pyelography
   b. Ultrasonography
   c. 24 hour cortisol level (Cushings disease)
   d. Catacholamines (Phcochromocytoma)
   e. CT Scan.

E. Medical Treatment :

1. Salt restricted diet
2. β-Blockcr: Atenolol, 50-100mg/day
3. Calcium Channel blocker : Nifidepine-10- 20mg four times a day.
4. ACE inhibitor : Enalapril 2.5 to 10mg/day
5. Diuretics : Frusemine 40mg/day
6. α blockers : Prazocin- 1 to 2mg/day
7. Clonidine/Methyldopa.

F. Surgical treatment:

1. Surgery for phcochromocytoma
2. Dilatation of stenosed renal artery.

G. Prevention and control:

I. Primary prevention

1. Health promotion
   a. Health education to create awareness.
   b. Nutrition: Salt and fats to be controlled.

c. Physical exercise:-Regular exercise avoidance of obesity, 'Shavasana' and certain other yogasanas are found to be useful.
d. Mental stress: Avoidance or mental stress. Meditation and certain other yogasanas are found to be userul.
e. Lifestyle modification.

II. Secondary prevention

a. *Early diagnosis*
Surveys or high risk population namely those who have the above mentioned risk factors as part of their life styles.

b. *Prompt and effective treatment:*
Treatment as outlined earlier with regular periodic check up and follow up visits.

III. Tertiary prevention

Rehabilitation
1. Physical rehabilitation and
2. Occupational rehabilitation may be required in serious cases.

3. Diabetes

A. Statistics:

Diabetes is one of the leading causes of death in the developed countries. The prevalence of diabetes has shown a rapid upward trend in the developing countries. There are an estimated 30 million cases all over the world.

In India it is seen that the prevalence is around 2-3 percent of the adult population.

B. Epidemiology:

I. Host factors which you should look for and highlight in the patient's history include.
   a. Age: Usually begins to manifest above 40yr. in MOD. Starts early around 10 yr. in JOD.
   b. Sex: equal in both sexes.
   c. Sedentary life style.
   d. PEM in early infancy and childhood.
   e. Viral infections eg. mumps and cocksackie B4.
   f. Physical stress, surgery, trauma, pregnancy.
   g. Family history.
   h. Obesity.

II. Environmental and Social factors:

The previous belief that Diabetes is a disease of the rich is no longer true. The previous information was also in all probability due to higher awareness and treatment seeking behaviour in the upper socio economic classes, leading to higher detection rates. The disease has almost equal prevalence in all crossections of the society.

C. Causes:

I. Primary (Idiopathic) Diabetes
1. Insulin Dependent Diabetes Mellitus (IDDM).
2. Non-insulin Dependent Diabetes Mellitus (NIDDM).

II. Secondary
1. Pancreatic disease.
   a. Pancreatitis
   b. Post pancreatectomy
   c. Pancreatic tumour
2. Endocrine disorders
   a. Cushing's Syndrome
   b. Glucagonomas
   c. Acromegaly
   d. Hyperthyroidism
3. Liver disorders
4. Drug induced
   a. Thiazide
   b. Steroids
   c. Diazoxide
   d. Streptozotoein

D. Clinical features:
1. Polyuria
2. Polydipsia
3. Polyphagia
4. Weight loss
5. Repeated infections like skin infections, urinary infections and others
6. Fatigue

E. Complications:
1. Diabetic Ketoacidosis.
2. Hyper- Osmolar Hyperglycemic Non-Ketotic Coma.
3. Lactic Acidosis.
4. Diabetic Retinopathy.
5. Diabetic Neuropathy.
6. Nephropathy.
7. Dermopathy.
8. Fungal infections.

F. Investigations:
1. Urine sugar and ketone bodies.
2. Fasting and post-prandial blood sugar.
3. Glucose Tolerance Test (G. T. T)
4. Haemoglobin A_1C or Glycated haemoglobin

G. Treatment:

I. Diet:
a) It should be palatable.
b) Diet exchanges must be taught to the patient
c) Diet pattern should be as physiological as possible
d) Small quantity of food with increased frequency
e) Body weight should be maintained to near normal.
f) Please look at diabetic diet on page shown later.

II. **Exercise:** Regular exercise or walking will help in maintaining blood sugar.

III. **Oral hypoglycemic agents:**

1. Sulphonyluria:
   - i) Chlorpropamide 100 to 500 mg/day
   - ii) Tolbutamide 500 to 3000 mg/day
   - iii) Glibenelamide 5 to 20 mg/day
   - iv) Glipizide 2.5 to 40 mg/day
   - v) Gliclazide 80-320 mg/day

2. Biguanide:
   - i) Phenformin 25 to 100 mg/day
   - ii) Metformin 500 mg to 2 gms/day

IV. **Insulin:**

a. Crystalline Insulin
b. Lente Insulin.
c. Human insulin -
   - i) Actrapid-HM
   - ii) Monotard-HM

d. Monocomponent -
   - i) Actrapid-MC
   - ii) Monotard-MC

V. **Treatment of Complications like.**

Neuropathy - i) B-complex
ii) Carbamazepine or Phenytoin for neuropathic pain
iii) Control of diabetes

Retinopathy - i) Photocoagulation
ii) Pituitary ablation
iii) Vitrectomy
iv) Control of diabetes.

H. Prevention and Control

I. Primary prevention: Health education related to
- a) Healthy nutrition, (high fibre, low sugar)
- b) Physical exercise

II. **Secondary prevention**

1. Early diagnosis: (screening) of high risk populations, e.g.
   - a) Persons above 40yrs.
   - b) Obese persons
   - c) Those with a family history
   - d) Women who deliver large babies.

   The screening tests are described above.
2. Prompt and effective treatment:
   - a) Treatment as outlined above with both, drugs and diet control.
   - b) Regular routine checkups of blood and urine.
   - c) Watch for complications
   - d) Patient Identity Card for action in emergencies.

III. **Tertiary prevention**

1. Disability limitation: By regular checkups and treatment; to avoid disabling complications, like :
   - a) Gangrene and Amputation
   - b) Kidney failure
   - c) Blindness.
2. Rehabilitation:
   - a) Physical rehabilitation: Reconstructive surgery, prosthesis.
   - b) Occupational rehabilitation, as per handicap.
   - c) Psychological rehabilitation of patients who have developed complications and become disabled or handicapped, to learn to live with the realities of life.

4. Obesity

Definition :

It is defined in terms of the Body Mass Index as above 30 for males and above 28.6 females.

A. Statistics:

More prevalant in developed countries than in developing countries.

Most studies in developed countries show a prevalence rate or 25% in adults and 15% in children. Figures for developing countries are not available, however it is estimated that about 10% of the adults and 2% children are obese in India.

B. Epidemiological factors:

Agent factors : Due to hypertrophy and hyperplasia of adipose tissue.

Host, social and Environmental factors : Look for the following factors in the patients history.

I. Age and Sex:

1. Obesity since childhood occurs in 30% of the cases.
2. In adult females, after pregnancy and at menopausal age.
3. In adult males beyond 40 yrs.

II. Familial tendency:

1. Due to rich dietary patterns.
2. Similiar lifestyles among family members.
3. Genetic factor.
4. Overeating associated with pregnancy and lactation, in well to do families.

III. Socio economic factors:

1. Directly proportional to the per capita income.
2. Considered as sign or prosperity in traditional North Indian families.

IV. Individual eating habits:

1. Preference for energy rich diets in marked excess to the daily requirement.
2. Frequent eating.
3. Alcohol consumption.
4. Psychological overeating as a result of anxiety neurosis, depression etc.
5. Junk food.

V. Physical exercise : Enquire about

1. Nature of job (physical activity involved)
2. Time devoted to sports or other physical activily.
3. Recent illness, particularly those which lead to long term restriction of physical activity e.g. fracture.

VI. Endocrine disorders:

1. Cushing's syndrome.
2. Cretinism, and Hypothyroidism.
3. Pituitary disorders.
4. Maturity onset Diabetes mellitus (MOD).
5. Insulinoma.
6. Hypothalamic disorders.

VII. Drug intake: Look for the history of consumption of the following drugs.

1. Corticostcroids.
2. Oestrogens.

Some indicators that are commonly used to measure obesity.

1. Broca's index :

The individual's height in cms minus 100 = max. wt. of the individual in kg.

e.g. For a person with a height of 182 cms. 182 - 100 = 82 Kg. is the max. permissible wt. Anything in excess of 82 Kg. will make the person to be considered as obese.

2. Body Mass Index (Quetelet's index) :

$$BMI = \frac{\text{weight (in Kg)}}{[\text{Height (in meters)}]^2}$$

Males:	a)	20 - 25	desirable range
	b)	22	desirable ideal
	c)	>30	obese.
Females:	a)	19 - 24	desirable range
	b)	21	desirable ideal
	e)	> 28.6	obese.

3. Corpulence index:

$$CI = \frac{\text{Actual Wt. (in Kg)}}{\text{Desirable Wt (in Kg)}}$$

in obesity this should exceed 1.2.

4. Ponderal index:

$$PI = \frac{\text{Ht (in cms)}}{\sqrt[3]{\text{Wt. (in Kg)}}}$$

5. Lorentz Formula:
   LF = [Ht (in cms)-100]– $\left[\frac{\text{Ht in (cms)-150}}{4}\right]$ (males)

   LF = [Ht (in cms)- 100]– $\left[\frac{\text{Ht (in cms)-150}}{2}\right]$ (females)

6. Fat fold thickness (skin fold thickness) :
   The fat fold thickness is measured using skin calipers at the following sites, Mid-triceps, Biceps, Subscapular and Suprailiac region. The sum total of the above measurements from all four sites should not be more than 40mm for adult males and 50mm for adult females. Normograms showing the ideal for males and females at various ages are available.
7. The total body mass is also calculated by weight/volume studies using water displacement technique.

C. Clinical features:

1. Weight gain
2. Buffalo hump
3. Moon face
4. Prone to develop
   a. Diabetes
   b. Hyper-lipidemia
   c. Gall stones

Complications :

1. Respiratory
   a. Pickwickian Syndrome
2. Cardio-vascular
   a. Hypertension
   b. Cor-pulmonale
   c. Varicose Veins
3. Gastro-intestinal
   a. Hiatus hernia
   b. Fatty liver
   c. Gall stones
4. Musculoskeletal
   a. Osteoarthritis
   b. Sciatica
   c. Flat foot
5. Miscellaneous
   a. Hernia
   b. Intertriginous dermatitis

D. Treatment:

1. Treatment of the secondary causes, like Hypothyroidism, Cushing's etc.
2. Diet:
   a. Restrict the calorie intake.
   b. Excessive eating should be avoided particularly at night.
   c. Small and frequent meals should be preferred.
   d. High roughage diet (which will have less calories) is preferred.
3. Exercise: It selectively decreases the body fat, while preserving the lean body mass.
4. Behavior Modification: It is advisable to treat abnormal patterns of eating behavior.
5. Drugs:
   a. Fenfluramine: 20mg/day
   b. Diethylpropion: 25mg thrice a day
   c. Biguanides
   d. Thyroid extract.
   c. Amphetamine.
6. Other methods:
   a. Body massage
   b. Steam-bath (sauna)
7. Surgery:
   a. Gastric pouch by Gastroplasty.
   b. Jejunoileal Shunt.
   c. Gastric balloon: Balloon is introduced through Gastroscope and kept inflated so that the patient gets a sense of satiety (feeling of fullness) after a small feed.
   d. Lipectomy: Removal of omental fat by laparotomy or liposuction.

E. Prevention and Control:

I. Primordial prevention:

1. Avoidance of lifestyles leading to obesity and inculcation of habits for a healthy life style, e.g.
   a. Dietary control.
   b. Physical exercise.

II. Secondary Prevention:

1. Identification of at risk individuals eg.
   a. Obese children
   b. Those with familial tedencies and starting them on obesity management regimes at an early age.
2. Treatment as outlined above.

III. Tertiary prevention:

1. Physical rehabilitation e.g. physiotherapy.
2. Occupational rehabilitation.

5. Rheumatic heart disease

A. Statistics:

Rheumatic fever and heart disease were fairly prevalent all over the world. However the last two decades have seen its rapid decline in the developed countries. It continues to be a public health problem where ever over-crowding and poor living conditions exist. ICMR studies indicate a prevalence rate of 2-6% in Indian school children.

B. Epidemiology:

I. Agent:

Although the causative agents is (Gr.-A β-Streptococcus) which is responsible for sore throat, the disease is due to an immunological process and hence we cannot talk in terms of incubation period, source of infection, mode of transmission, period of communicability and secondary attack rate of the disease.

The source of Gr. A β-Streptococcal infections are pharyngeal carriers.

The mode of transmission of the streptococcus is through air by droplet infection.

II. Host factors:

1. Age: 5-15 yrs.
2. Sex: Both sexes are equally affected.
3. Immunity: RHD and RF is an immune response to Gr. A β-streptococci throat infections.

III. Environmental and Social factors

1. Overcrowding.
2. Poor ventilation.
3. Poor housing conditions.(e.g. slum dwellers).
4. Poor socio economic status.
5. Poor health care facilities (particularly school health facilities).

C. Clinical features:

1. Asymptomatic, detected on routine examination
2. Exertional dyspnoea
3. Hemoptysis
4. Palpitation
5. Repeated respiratory tract infections
6. Symptoms of cardiac failure

D. Inyestigations :

1. Anaemia - Normochromic and Normocytic
2. Leukocytosis - if associated with Rheumatic fever or infective endocarditis.
3. Urine examination for microscopic hematuria (Infective endocarditis)
4. Electrocardiogram:
   a. For chamber enlargement.
   b. For Rhythm disturbance.
5. X-ray chest PA :
   a. Chamber enlargement
   b. Chest infection
   c. Pulmonary oedema.
6. ASLO titre for Rheumatic fever.
7. Blood culture to rule out infective endocarditis.
8. Echocardiography to see
   a. Vegetations
   b. Extent of Valve involvement
   c. Presence of clots
   d. Ejection fraction
9. Cardiac Cathetcrisation for pressure changes in various chambers of the heart.

E. Medical Treatment:

1. Salt and Water restriction.
2. Diureties like frusemide (40mg) once a day. with Potassium replacement
3. Digoxin: lt is indicated in
   a. Atrial fibrillation
   b. Cardiac failure.
4. Rheumatic prophylaxis: Inj. Penidura 1.2 mega unit IM once in three weeks
5. Prophylaxis against Infective endocarditis. Proper antibiotics 1/2 an hour before and 2 days after procedures or operations.

F. Surgery :

Indications:

1. Symptomatic patient with Class III - IV dysponea
2. Pulmonary oedema
3. Low output state
4. Systemic embolisation

Types of Surgery:

1. Valvuloplasty.
2. Valve replacement.

G. Prevention and Control:

I. Primary Prevention:

1. Health promotion: Health education regarding RF, RHD to school children, teachers and parents to get sore throat treated.
2. Specific protection: Treating sore throat with Penicillin and Amoxicillin.

II. Secondary Prevention:

1. Early diagnosis.
   a. Surveillance of school children for RF, RHD.
   b. Throat swabs for detection of Gr. A β streptococci, where such facilities are available.
2. Prompt and effective treatment:
   For patients diagnosed as RF, treatment with penicillin periodically for atleast 5yrs, or till the child reaches 18yrs of age, which ever is later.

III. Tertiary Prevention:

1. Disability limitation: Cardiac surgery
2. Rehabilitation
   a. Physical rehabilitation.
   b. Occupational rehabilitation.
   c. Psychological rehabilitation may be required in long standing cases after cardiac surgery.

6. Tuberculosis

A. Statistics

World :

1. Prevalence of infection (tuberculin positivity) is around 2-3% for developed countries and 50- 75% for developing countries.
2. Prevalence of cases (sputum positives) is almost nil in developed countries and 2.5 to 5 per 1000 population in developing countries.

India:

1.	Incidence of infection	3%
2.	Prevalence of infection	40%
3.	Incidence of sputum positives	0.25%
4.	Prevalence of sputum positives	1 %

B. Epidemiology:

I. Agent: (Mycobacterium Tuberculosis)

1. *Source of infection*: Human:- coughing, spitting [Look for a case in the family or close contacts]
   Bovine: milk, meat. (rare in India)
2. *Mode of transmission:* : Air borne: Droplet infection, droplet nuclei. (not through fomites)
3. *Period of communicability* : As long as the patient remains sputum +ve.
4. *Incubation period:* : Long (4 wks to years)

II. Host: Look for the following:

1. *Age:* Prevalence increases with age maximum incidence is between 5-20 yrs.
2. *Sex:* Male: Female ratio is 5:1
3. *Nutritional status:* Seen more in the malnourished
4. *Immunization:* Ask for the history of BCG vaccination and look for BCG scar on the left shoulder

III. Environmental and Social factors: Look for,

1. Overcrowding and poor ventilation.
2. Low Socio economic class.
3. Poor facilities for waste disposal.

C. Clinical features:

I. Symptoms

1. Sometimes asymptomatic - detected on routine check-up.
2. Anorexia, malaise, weakness and undue fatigue.
3. Evening rise of fever, specially low grade fever.
4. Cough with or without expectoration.
5. Haemoptysis in later stages.
6. Pleuritic pain.
7. Dyspnoea.

II. Signs:

1. Thin, cachectic patient
2. Post-tussive crepts, specially in the apical region
3. Signs of cavity (cavernous/bronchial breathing)
4. Signs of fibrosis (mediastinum shifted to same side, dullness)
5. Signs of pleural effusion (mediastinal shift to opposite side and dull note, with diminished vocal resonance)
6. Signs of hydropneumothorax (shifting dullness and succussion splash)

Complications

1. Massive haemoptysis
2. Malnutrition
3. Bronchiectesis
4. Amyloidosis
5. Scar carcinoma

D. Investigations:

1. CBC with ESR will show,
   a. Anaemia
   b. Leukocytosis
   c. Raised ESR
2. X-Ray chest
   a. PA view
   b. Lordotic view for apical Koch's
   c. Lateral decubitus view for minimal pleural effusion
3. Sputum AFB
4. Tuberculin test
5. Tuberculous antigen and antibody in fluid and serum.

E. Treatment :

Anti-Tuberculosis Chemotherapy Regimes

Conventional

1.	Streptomycin INH Thiacetazone	2 months followed by	INH Thiacetazone	10 mths.
2.	Streptomycin INH	2 months. followed by	INH (twice weekly) streptomycin (twice weekly)	10 mths.
3.	Thiacetazone INH	18 months.		

Short-term

1.	Rifampicin Streptomycin INH Pyrazinamide	2 months followed by	Rifampicin INH	4 mths.
2.	Rifampicin Streptomycin INH Pyrazinamide	2 months followed by	Rifampicin (twice weekly) INH (twice weekly)	4 mths.
3.	Rifampicin INH Pyrazinamide	2 months followed by	Rifampicin INH	4 mths.
4.	Rifampicin Streptomycin INH Pyrazinamide	2 month followed	Streptomycin (twice weekly) INH (twice weekly) Pyrazinamide (twice weekly)	5 mths.
5.	Rifampicin INH Streptomycin Pyrazinamide	2 months followed by	INH Thiacetazone	6 mths.
6.	Rifampicin Streptomycin INH Pyrazinamide	(thrice weekly) (thrice weekly) (thrice weekly) (thrice weekly)	6 months.	

F. Prevention and Control:

As per the WHO, T.B control is, to bring the infection rate in the 0-14 age group to 1% level (currently about 40% in India)

I. Primary prevention:

1. *Health promotion* : Health education to patient's contacts and general public about
   a. Mode of transmission
   b. Disposal of sputum and infective material
   c. Methods to prevent spread from patients to healthy individuals
   d. Regularity of treatment and follow-up
2. *Specific protection* :
   a. Immunoprophylaxis: BCG Vaccination which is a part of the UIP program is now believed to protect only against severe forms of T.B e.g. T.B. meningitis and miliary T .B.
   b. Chemoprophylaxis: The utility of INH as a prophylactic measure can be considered only for children upto 6 yrs of age, who are in close contact with sputum +ve cases.

II. Secondary Prevention:

1. *Early diagnosis:*
   a. All patients with the following symptoms
      i Cough for more than 4 wks.
      ii Low grade, evening rise of fever and night sweats.
      iii Chest pain.
      iv Haemoptysis.
      should be investigated for T.B.
   b. Contact tracing measures, for family members and other close contacts of cases.
   c. Mass population surveys, in areas where an extremely high prevalence is suspected
   d. Mass miniature radiography and tuberculin testing have no value as case detection measures.
2. *Prompt and effective treatment:*
   a. Domiciliary treatment as above.
   b. Regular treatment and follow up.
   c. Defaulter tracing.

III. Tertiary Prevention:

1. *Disability limitation:* Can be achieved through early diagnosis, prompt and effective treatment and regular follow up.
2. *Rehabilitation* :
   a. Occupational rehabilitation may be required in certain cases, due to decreased pulmonary functions.
   b. Psychological rehabilitation of the cured patient, in view of the social stigma and the false beliefs associated with the disease.

7. Chicken Pox

A. Statistics : .

Occurs world wide, however, it is not considered as a serious public health problem and hence no statistics are available.

B. Epidemiology :

I. Agent : (Varicella - Zoster Virus)

1. Source of infection:
   The secretions from the respiratory tract of the patients.
   The vesicle fluid in the first few days.
   Scabs are not infective.
2. Mode of transmission:
   Droplet infection, through air.
   Direct contact.
   Rarely, fomites.
   Transplacental.
3. Period of communicability:
   2 days before the rash commences.
   and 5 days after the rash commences.
4. Incubation period:
   7-21 days, average 15 days.
5. Secondary attack rate: (SAR)
   Highly contagious. SAR is around 90% in susceptible contacts.

Enquire about other cases in the ncighbourhood.

II. Host:

1. Age: More common in children and young persons, but more severe in adults. One attack gives life long immunity.
2. Sex: Equal in both sexes.

III. EnvironInental and Social factors:

1. Seasonal; mainly during spring and summer in India.
2. Over crowding and poor ventilation.
3. Persons from southern states eg. Kerala Karnataka are usually not exposed in their childhood and get it at a later age when they migrate to the other states for jobs (eg. nurses from Kerala, hotel boys from Mangalore).

C. Clinical features:

I. Prodromal Stage:

1. Headache
2. Sore throat
3. Fever: continuous and lasts for 3-5 days.

II. Stage of Erruption :

1. Enanthema: Earliest lesion is in the buccal and pharyngeal mucus membranes.
2. Exanthema:
   a. It occurs first on the back and then over the chest and abdomen
   b. Macules papules vesicles occur in rapid evolution.
   e. Pruritus and generalised lymphnode enlargement occurs.

D. Complications:

1. Secondary bacterial infection of the skin.
2. Pneumonia.
3. Encephalitis.
4. Thrombocytopenia.
5. Congenital abnormalities, if it occurs in the first trimester of pregnancy.

Following congenital abnormalities are noticed,

1. Microcephaly
2. Ocular damage
3. Atrophied limbs

E. Medical treatment:

1. Bed rest.
2. Calamine lotion, and anti-histaminics for pruritus.
3. Antibiotics for infection.
4. Corticostcroids for encephalitis.
5. Anti-viral agents.
   a. Adenosine Arabinoside.
   b. Acyclovir.

F. Prevention and Control:

This disease has no serious consequences and hence is not considered a serious public health problem.

However as primary prevention, specific protection using varicella Ig or Zoster-Immune Plasma is used for contacts with serious immuno-deficiency.

Varicella Zoster vaccine although successfully tried, is not considered a cost effective proposition either for mass prophylaxis or commercial distribution.

8. Mumps

A. Statistics:

Mumps occurs world wide but since it is not of serious public health consequences, there are no available statistics.

B. Epidemiology:

I. Agent: (Myxovirus)

1. Source of infection: Oral and Respiratory tract secretions of clinical and subclinical cases.
2. Mode of transmission:
   a. Droplet infection through air.
   b. Kissing.
   c. Rarely through fomites.
3. Period of communicability: One week before the onset of clinical features and one week after the onset.
4. Incubation period. 15-20 days.
5. Secondary attack rate: About 90%.

II. Host:

1. Age: Maximum incidence during 5-15yrs.
2. Sex: Slightly more in males.

III. Environmental and Social factors:

1. Mainly in winter and spring.
2. Over crowding and poor ventilation.
3. Low socio-economic living styles.

C. Clinical features:

I. Prodromal phase:

Malaise
Anorexia
Sore throat
Fever with chills

II. Parotitis: (Salivary adenitis)

1. The parotid gland enlarges progressively over 1 to 3 days.
2. Resolves after one week of maximum swelling.
3. Ear lobule is lifted.
4. Skin over the gland is neither warm nor erythematous.
5. Pain and tenderness are marked.
6. Reddening and pouting of the orifice of Stensen's duct.
7. Sub-maxillary and Sub-mandibular gland may be enlarged.
8. Parotitis is usually bilateral (66%).

III. Fever:

1. It rises upto 103° to 104°F.
2. It is intermittent or remittent.
3. It falls by lysis within one week.

Complications:

1. Epididymo-orchitis
   a. Incidence 20-35%
   b. It occurs 7 to 10 days after onset of parotitis.
   c. Bilateral in 3 to 17%.
2. Meningitis.
3. Oophritis.
4. Acute Pancreatitis, It occurs in 2nd week.
5. Glomerulonephritis.
6. Thyroiditis.

D. Investigations:

1. Leukopenia with lymphocytosis. Leukocy tosis occurs if orchitis occurs.
2. C.S.F. shows pleocytosis in meningitis.
3. ELISA and Immunoflourscent test used to detect viral antigen from blood, saliva and secretions from Stensen's duct.

E. Treatment:

1. Bed rest.
2. Oral hygicne: Mouth wash with chlorhexidine solution or $KMnO_4$ solution (1:5000)
3. Analgesics: Paracetamol or Aspirin to control pain and fever.
4. Managemcnt of orchitis
   a. Complete bed rest.
   b. Support to the scrotum.
   c. Analgesics.
   d. Antibiotics.
   e. Steroids if required.
   f. Exeruciating pain is treated by :
      i) Incision of the Tunica Vaginalis
      ii) Injection of xylocaine (1%) to be injected in the spermatic cord at the external inguinal ring level.

F. Prevention and Control:

I. Primary prevention:

1. **Immunoprophylaxis:**
   a. Live attenuated vaccine, single dose, 0.5ml subcutaneously or intramuscularly, is given separately or in combination with Measles and Rubella vaccine between 12 and 15 months of age, but can be given later also.
   b. Ig can be given to high-risk contacts with immune suppression.
2. Chemoprophylaxis : No known chemoprophylaxis is available.

II. Secondary prevention:

1. Early diagnosis: As advised above.
2. Prompt and effective treatment: As outlined above. Watch for complications like orchitis. Follow up to check for sequcle like diabetes.

III. Tertiary Prevention:

Has no role in this disease.

9. Measles

A. Statistics:

Most of the developed countries have reported near elimination of measles. In the developing countries it is a serious health problem among the under 6 yrs children, with high case fatality rates in the malnourished.

The annual incidence rates of measles in India are 0.2% for all ages and 1.5% for children under 6 yrs. of age. 10-12% case fatality rate among children under 6 yrs. is reported.

B. Epidemiology:

I. Agent: (Measles Virus - is a Paramyxovirus)

1. Source of infection : Oro-pharyngeal and respiratory tract secretions of measles cases. (sub clinical cases and carriers are nonexistent in this disease)
2. Mode of transmission: Droplet infection and droplet nuclei.
   Rarely through infection of conjuntiva. Vaccine virus is not transmitted by this mode of transmission.
3. Period of communicability: A week before and a week after the appearance of the rash.

II. Host:

1. Age: 80% of the cases occur between 6 months and 6yrs. of age. Measles in adults is also seen.
2. Sex : Has equal incidence in both sexes.
3. Nutritional status of the host plays an important role in the severity of the disease, with malnutrition causing high mortality.
   Following an attack of measles, the child slips into further degrees of malnutrition giving way to other infections, most commonly tubcrculosis. This disease typically prcscnts thc vicious cycle of Infection Malnutrition.

III. Environmental and Social factors:

1. Seen more in winter.
2. Over crowding and poor ventilation
3. Poor socio - economic living conditions.

C. Clinical features:

I. Prodromal Stage : It lasts for 3 to 4 days.

1. Fever:
   a. It may be as high as 105°F
   b. Continuous.
   c. Without rigor.
2. Malaise, irritability, conjunctivitis photophobia.
3. Moderately severe hacking cough.
4. Koplik's spots,
   a. They appear 1 to 2 days before the onset of the rash.
   b. They are present on the mucus membranes of the mouth and occasionally on the conjuctivae or intestinal mucosa.
   e. They are small, red, irregular lesions with blue-white centers with reddish mottled areola around them.
   d. They fade out with the appearence of the rash.
5. Laryngeal stridor or hoarseness of voice.

II. Eruptive stage:

1. Skin rash:
   a. It appears on the 5th day
   b. Red macules appear, first over the forehead and behind the ears and then spread downwards over the face, neck and the trunk.
   c. Lesions persist for 3days and disappear in the same order in which they appear.
2. Mucus membrane: Conjunctivitis, laryngitis and bronchitis can occur.

III. Defervescence stage:

1. Patient becomes afebrile.
2. Rash fades out from above downwards.
3. General well being improves.

Complications:

1. Bronchitis, Bronchiolitis or Pneumonia Secondary bacterial infections arc common
2. Corneal ulcer: This may follow severe conjunctivitis.
3. Myocarditis and pulmonary oedema.
4. Encephalitis: Occurs in 1 out of 1000 cases.
5. Hepatitis.
6. Acute Glomerulonephritis.

7. Late complications.
   a. Bronchiectasis.
   b. Sub-acute Sclerosing Pan Encephalitis (SSPE). It occurs about 7 years after an attack of measles. It is one of the rare but fatal complications.

D. Investigations:

1. Leukopenia specially lymphopenia.
2. Cell culture inoculation of virus contaminated secretions.
3. Complement fixation, Enzyme Immunoassay and haemagglutination inhibition test.
4. CSF Examination in Viral Encephalitis.

E. Medical Treatment:

1. Bed rest during fever
2. Symptomatic treatment
   a. Fever - Paracetamol
   b. Cough - Linctus codeine
3. Antibiotics for bacterial infections.

F. Prevention and Control:

I. Primary prevention:

1. Health Promotion: Health education towards improvement in nutritional status, and the need for immunization.
2. Specific Protection :
   a. Immunoprophylaxis: Live attenuated vaccine (Schwarz strain) given 0.5ml subcutaneously after 9 months of age gives life long immunity. Included in the UIP. Inview of measles now freqently affecting children below 9 months in developing countries, the Edmonton Zagreb Strain would be preferable for use in India.
   b. Chemoprophylaxis: Has no role in this disease.

II. Secondary prevention:

1. Early diagnosis: Using methods mentioned above
2. Prompt and effective treatment: As outlined above. Follow-up for nutritional deficiency diseases mainly PEM and other sequelae.

III. Tertiary prevention:

Is aimed at nutritional rehabilitation of children who have become severely malnourished as a sequelae of measles.

10. Leprosy

A. Statistics:

Leprosy is a disease that finds a mention in almost all ancient literature. It has a world wide occurrence. It has declined rapidly in the last century in the developed nations. It however, continues to be a public health problem in the developing countries of Asia, Africa and the Western Pacific. India has almost 1/3 of the leprosy cases in the world, with a prevalence ratio of 5- 7 per 1000 population. India has 4 million cases. 2 lakh cases are either cured or die while 2 lakh new cases are reported each year. Tamil Nadu, Bihar, Andhra Pradesh, Pondicherry and Lakshadweep are highly endemic areas.

B. Epidemiology:

I. Agent: (Mycobacterium Leprae.)

1. *Source of infection* : Lepromatous and Borderline Lepromatous cases. Look for a case in the family or among close contacts.
2. *Mode of transmission* :
   a. Droplet infection
   b. Contact transmission.
3. *Period of communicability* : Lepromatous and Borderline cases become non infectious within 2 weeks of treatment with *Rifampicin* or 3 months of treatment with *Dapsone.*
4. *Incubation Period :* Long, usually 3 to 5 yrs. Shorter incubation in *tuberculoid* leprosy.
5. *Secondary, Attack rate:* 5-12%

II. Host :

Look for :

1. Age : Occurs at all ages, with maximum incidence during 10 to 20 years.
2. Sex: More in males.

III. Environmental and Social Factors :

Look for :

1. Low socio-economic class.
2. Overcrowding and poor ventilation
3. Presence of humidity in the atmosphere favours the transmission of leprosy.

C. Clinical Features:

Presentation depend upon cell mediated immunity of the patient.

Immunological status	Type of Disease
1. Poor immunity	Lepromatous (LL) Leprosy
2. Intermediate immunity	Borderline Leprosy (BB,BL,BT)
3. Good immunity	Tuberculoid (TT) leprosy

Lepromatous Leprosy.

Early manifestations commonly missed by patients include:

1. Nasal symptoms:
   a. Stuffiness
   b. Crust formation
   c. Blood stained discharged from nose.
2. Oedema of legs and ankles
   a. Bilateral and symmetrical
   b. More marked in the evening.

Other overt manifestations include:

1. Skin lesions
   a. Macules (more common), papules and *nodules.*
   b. Bilateral symmetrical and large.
   c. Sites: face, arms, buttocks, legs and trunk.
   d. Hypopigmented.
2. Leonine facies
   a. Thickening of skin of forehead, causes deepening of the natural lines.
   b. Ear lobes are thickened
   c. Eyebrows are lost (madarosis)
   d. Nose may collapse
3. Superficial Nerves
   a. Thickened
   b. Glove and Stocking anesthesia
4. Testicular atrophy leading to
   a. Sterility
   b. Impotence
   c. Gynecomastia.
5. Bone
   a. Periostitis
   b. Disuse Osteoporosis
6. Eye
   a. Superficial Punctate Keratitis

Tuberculoid Leprosy:

1. Nerve
   a. It produces mononeuritis (single nerve affection) and mono-neuritis multiplex (multiple nerve involvement).
   b. Thickened nodular and palpable nerves.
   c. Common nerves involved are as follows.

Nerve involved	Site
1. Greater auricular	Neck
2. Ulnar	Above medial condyle
3. Median	Carpal tunnel
4. Sural and Superficial peroneal	Lower leg.
5. Posterial tibial	Behind lateral malleolous

30% of nerve fibres should be destroyed to produce sensory signs and symptoms.

2. Skin:
   a. Macules and papules
      - Assymetrical
      - Large
      - Hypopigmented with anaesthesia
   b. Site: Lateral aspect of arms, buttocks, legs and shoulders

Borderline - Leprosy (BB)

It occurs with intermediate degree of resistance. It can be

i) Borderline (BB)
ii) Borderline lepromatous (BL)
iii) Borderline Tuberculoid (BT)

These types depend upon immunity, and may change to Lepromatous (if immunity decreases) or Tuberculoid (if immunity improves).

D. Investigations

1. Anaemia with raised ESR (Normochromic and normocytic)
2. Skin smear for AFB
   a. Scraping
   b. Incision
3. Nasal Scraping from inferior turbinate for AFB
4. Skin biopsy
5. Nerve biopsy
6. Histamine test.

One drop of histamine 1:1000 dilution is placed on the area of the skin to be tested and another on the control site. A superficial prick is made through the drop.

Result		Interpretation
Bright flare	—	Normal skin
Delayed and feeble flare	—	Indeterminate (BB)
Absent	—	Tuberculoid (TT)

Differentiation of various types of Leprosy.

Characteristic	TT	BT	BB	BL	LL
1. Number of lesion	Single	Single or few	Several	Many	Plenty
2. Size of lesion	Variable (may be large)	Variable	Variable	Variable	Small
3. Surface of lesion	Dry & Scaly	Dry	Dry/Shiny	Shiny	Shiny
4. Anaesthesia	Present	Slight	Slight	Slight	Absent
5. Hair loss in lesion	Present	Present	Slightly present	Slightly present.	Absent

7. Sweat test:

0.2 ml of 1:1000 solution of pilocarpine is injected intradermally. Paint with iodine and dust with starch powder.

Result		Interpretation
Blue discolouration	-	Normal skin
No colour	-	leprosy

Quinizarin powder can be used in place of starch and iodine.

8. Lepromin test:

It is used to classify the type of lesion.
0.1 ml of antigen is injectcd intra-dermally.
Test results are read after 4-5 weeks.

Utility of Lepromin test

Test	Type of leprosy
+++	TT
++	BT
- ve	BB, BL, LL

Interpretation	Observation
Negative	No Wheal
Doubtful	A papule 3 mm or less in diameter
+	Papule 4 to 6 mm
++	Papule 7 to 1.0 mm
+++	> 1.0 mm or ulceration.

Lepra Reaction	Type I (Reversal reaction)	Type II (Erythema nodosum leprosum)
1. Clinical features	a. Existing lesions become erythematous oedematous. b. New skin lesions may develop. c. Neuritis d. Constitutional symptoms like fever and malaise are unusual.	a. Multiple subcutaneous tender and inflammed nodules come up. b. Each nodule lasts for a week or two followed by new crops of nodules c. Neuritis d. Systemic disturbances. i. Fever, malaise, lymphadenopathy and arthritis are common ii. Acute glomerulonephritis iii. Iritis iv. Epistaxis
2. Affected variety	It occurs in BB, BT and BL	It occurs in BL and LL
3. Immunological	It is due to type IV or delayed hypersensitivity	It is due to type III or Arthus reaction
4. Haematological findings	None characteristic	a. Normocytic, normochromic anaemia b. Polymorphonuclear leukocytosis c. Raised ESR d. Thrombocytosis e. Raised IgG, IgM, C2 and C3
5. Histology	Few bacilli with lymphocytes, macrophages, epitheloid and giant cells infiltration	Fragmented, granular bacilli Neutrophillic infilitration and vasculitis.
6. Treatment	a. Analgesics b. Steroids in case of neuritis.	a. Analgesics b. Antipyretics c. Prednisolone 60-120 mgs/day in severe cases d. Clofazimine should be given. It has antiinflammatory action in addition to antileprosy action. e. Thalidomide, the most effective drug is given in a dose of 200 mg twice a day to males & nonpregnant females (it produces phecomelia if given to pregnant women). f. Chloroquine. g. Cytotoxic drugs.

E. Medical Management:

I. Multi-bacillary disease (LL, BL, BB)

1. Rifampicin - 600 mg once monthly
2. Dapsone - 100 mg daily
3. Clofazimine - 300 mg once monthly or 50 mg daily

If not tolerated as in light-skinned patients, give Ethionamide or Prothionamide 250-375 mg/ day.

Total duration: Minimum 2 years or more

II. Paucibacillary disease (TT, BT)

1. Rifampicin - 600 mg once monthly
2. Dapsone - 100 mg daily.

Total duration - 6 months.

Smears from six sites should be taken every six months for at least 3 years after stopping treatment to detect bacteriological relapse.

III. Treatment of acute Iritis :

1. Steroid eye drops.
2. Atropine eye ointment.

IV. Chronic leg Ulcer management :

1. Zinc skin ointment.
2. Regular dressing.

V. Orthopedic and Plastic Surgery :

Effects of Pregnancy on a female patient of leprosy.

I. Worsening of leprosy due to depression of cell mediated immunity
II. Chances of intercurrent infections are increased
III. Increased incidence of Lepra reaction.

Effects of Leprosy on foetus.

I. Low birth weight baby
II. Slow growth rate (I. U. G. R.)

F. Prevention and Control:

I. Primary Prevention:

1. *Health Promotion:* Health education, to create awareness about the disease, to protect self and others against the disease and cooperate with early detection measures.
2. *Specific Protection:*
   a. Immunoprophylaxis: Atleast two vaccines are under community (phase IV) trials namely:
      i) The ICRC vaccine (ICMR)
      ii) Convit's vaccine (WHO)

BCG vaccine is also said to provide some degree of immunity against leprosy.

b. Chemoprophylaxis: Although Dapsone and Acedapsone (long-acting) has been tried out in close contacts, its use for mass prophylaxis is of doubtful value.

II. Secondary Prevention:

1. Early diagnosis:
   Case detection: The following strategies are used.
   a. Mass (population) surveys: In suspected hyperendemic (more than 10 cases per 1000 population) areas.
   b. Surveys of high risk groups:
      i) Slums and low socioeconomic dwellings
      ii) Schools for low socioeconomic classes eg. municipal schools.
      iii) Labourers in industries.
   c. Contact survey: Examination of family members and other close contacts of Lepromatous and borderline Lepromatous cases.
   d. Health education: For secking treatment for any hypopigmented, anaesthetic patches.
2. Prompt and effective treatment:
   a. Multi drug therapy as shown here.
   b. Full and regular treatment.
   c. Maintenance of proper treatment records.
   d. Follow up of defaulters.

III. Tertiary Prevention:

1. Disability limitation: can be achieved through prompt, regular and effective treatment. 20% cases are said to develop residual deformities.

2. Rehabilitation:

a. **Physical rehabilitation:**
   i) Physiotherapy.
   ii) Reconstructive surgery and plastic surgery.
b. **Occupational rehabilitation:**
   Occupational therapy.
c. **Psychological rehabilitation:**
   To prepare the cured patient psychologically to face the world, in view of the social stigma attached to the disease.
d. Social rehabilitation: Education of the general public to accept the cured patients as normal.

11. Scabies

A. Statistics:

Scabies is fairly common among people who have poor personal hygeine, more so in children.

In the developed countries it is found in ghetto or slum children and in the developing countries among the low socioeconomic groups in both urban and rural areas and among school children.

No definite statistics in terms of incidence or prevalence are available.

B. Epidemiology:

I. Agent: Sarcoptes scabiei :

1. Source of infection: Infected patients and their clothes and linen.
2. Mode of transmission:
   a. Direct contact with patients.
   b. Fomites.
3. Period of communicability : Till such time as the infection persists in untreated cases, usually 4-6 wks.
   Patient becomes non-infective within 3 days of effective treatment.
4. Incubation period: Usually 7 days.
5. Secondary attack rate: Around 80% in children and around 30% in adult contacts.

II. Host:

1. Age: Maximum incidence in school going children, occurs at all ages.
2. Sex: Equal in both sexes.
3. Poor personal hygiene.

III. Environmental and Social factors:

1. Maximum incidence in winters.
2. Overcrowding
3. Low socioeconomic living style.
4. Limited availability of water for washing.

C. Clinical Features:

1. Severe itching, which is worse at night
2. Common with other family members
3. Burrow is the greyish, serpentine, dotted line on the skin which represents the tunnel made by the female mite.
4. Sites of Burrows are :
   a. Interdigital folds
   b. Flexor aspects of wrists
   c. Anterior axillary folds
   d. Umibilicus
   e. Lower abdomen
   f. Genitalias.
   i. Buttocks and thighs.

Complications :

1. Secondary infections
2. Urticaria and Eczema
3. Id Eruption
4. Glomerulonephritis.

Variants:

1. Scabies in clean and healthy person.
2. Scabies incognito:
   It occurs in a patient taking steroids.
3. Norwegian Scabies :
   It occurs in immuno-compromised persons.
4. Facial scabies in infants.

D. Medical Treatment:

Principle:

1. All family members are treated simultaneously.
2. Clothes should be boiled and kept in the sun.
3. Secondary infcctions should be treated.
4. All the drugs used locally should by applied below the neck on 3 consecutive days.

I. Drugs:

1. Benzyl Benzoate (25%)
2. Gamma benzenchexachloride
3. Sulphur (10%)
4. Mitigal (10%)
5. Monosulfiram.

II. **Control of pruritus with Anti-histaminic drugs:**
   1. Astemezole.
   2. Terfenadine.

III. **Antibiotics for treating secondary infections:**
   1. Penicillin.
   2. Ampicillin.
   3. Amoxicillin.
   4. Erythromycin.

E. Prevention and Control:

I. **Primary prevention:**
   I. Health promotion:
   Health education related to personal hygiene, daily washing of clothes and daily bathing.
   2. Specific protection : Nil

II. **Secondary prevention:**
   1. Early diagnosis: On the basis of symptoms described : above.
   2. Prompt and effective treatment
      a. As outlined above.
      b. Follow up for ensuring complete cure.
      c. Treatmcnt of all close contacts.

III. **Tertiary Prevention:** Has no role in this disease.

12. Sexually transmitted diseases

A. Statistics:

The true incidence of these diseases will never be known due to the secrecy involved in the treatment of these diseases. The over all prevalence of all the ST Diseases put together, in the vulnerable population, is estimated to be around 10 -12 percent.

Syphilis, Gonorrhea and Chancroid are prevalent all over India while LGV, and Donovanosis are reported more commonly from the southern states of India.

Syphillis and Gonorrhea are discussed here.

B. Epidemiology:

I. Agent: Syphillis-Treponema pallidum
Gonorrhoea - Neisseria gonorrheae.

1. Source of infection:
   a. Patients with primary chancre, condylomata or the mucocutaneous junctions of patients in the primary and secondary stage in syphilis.
   b. Untreated, symptomatic and asymptomatic cases in gonorrhea.
2. Mode of transmission:
   a. Sexual contact during primary and secondary stages in syphilis.
   b. Transplacental in syphilis
   c. Sexual contact in gonorrhea.
   d. Fomites in gonorrhea
   e. Direct instillation in the conjunctive as in opthalmia neonatorum.
3. Period of communicability:
   a. Through out the primary and secondary stages, some times as long as 4yrs. in syphilis.
   b. As long as the case remains untreated in gonorrhea.
4. Incubation period:
   a. 9 to 90 days, average 18 days, in syphilis
   b. 3 - 9 days in gonorrhea, average 4 days.

II. Host:

1. Age: Young adults show maximum incidence.
2. Sex: Higher in men, more severe in women.

III. Environmental and Social factors:

1. Illiteracy and ignorance.
2. Poverty and unemployment, prostitution.
3. Urbanisation and rapid industrialization.
4. Marital problems.
5. Sexual myths.
6. Peer pressures.
7. Changing moral values.
8. Travel and migration for business, employment and pleasure.

Syphilis :

C. Clinical features

Disease stage	Period
1. Primary	9 to 90 days
2. Secondary	After 12 weeks
3. Early latent	< less than 1 year
Late latent	more than 1 year
4. Tertiary	2 years.

Primary syphilis :

1. Primary chancre:
   Single painless papule which rapidly becomes eroded, indurated.
   Site: Penis, anal canal, rectum,within the mouth or on the external genitalia.
2. Regional Lymphadenopathy:
   Nodes are firm, non-suppurative and painless.

Secondary syphilis:

1. Localised or diffuse symmetric muco-cutaneous lesions.
   a. Bilateral symmetrical.
   b. Pale red or Pink.
   c. Non - pruritic.
   d. Discrete - round macules.
   e. Distributed on the trunk and proximal extremities.
2. Gondyloma lata: (Seen in 1.0% of the patients)
   a. It occurs in warm, moist and intertriginous areas.
   b. Broad, moist.
   c. Pink or greyish white.
   d. Highly contagious.

3. Constitutional symptoms:
   a. Fever (mild).
   b. Sore throat.
   c. Malaise.
   d. Headache.
4. G. T. symptoms:
   a. Linitis Plastica.
   b. Patchy proctitis.
   c. Lymphosarcoma of stomach (rare).
   d. Hepatitis.

Latent syphilis :

It is diagnosed by the finding of a positive specific treponemal antibody test for syphilis, together with a normal cerebrospinal fluid examination, absence of clinical manifestations of syphilis on physical examination and with the absence of history of primary or secondary lesion.

Late syphilis:

1. Aortitis, aortic aneurysm.
2. Asymptomatic neurosyphilis:
   No manifestations of neurosyphilis but CSF abnormalities secn.
3. Symptomatic Neurosyphilis:
   a. Meningovascular.
   b. General paresis of insane (GPI).
   c. Tabes dorsalis.

Late benign Syphilis: (Gumma)

a. Usually single, but may bc multiple.
b. Size varies from few mellimeters to several centimeters.
c. Site: skin, skeletal muscle, mouth and upper respiratory tract.

Congenital Syphilis:

Early
a. Within first 2 years of life.
b. Rhinitis.
c. Bullae, vesiclcs, superficial desquamation (Syphilis pemphigus).
d. Condylomata lata.
e. Osteochondritis and osteitis.
f. Hepatosplenomegaly with Lymphadenopathy.
g. Anaemia, Jaundice.

Late
If syphilis remains untreated after 2 years Hutchinson's triad is secn which includes
a. Interstitial Keratitis
b. Deafness
c. Hutchinson's teeth (centrally notched, widely spaced, peg shaped upper central incisors).

D. Investigations:

I. Using Non-treponemal antigen:
   1. Venereal Disease Research Laboratory (VDRL) - microscopic flocculation test.
   2. Rapid Plasma Reagin (RPR) - macroscopic flocculation test.

II. Using Treponema) antigen:
   1. Fluorescent treponemal antibody - absorption (FTA - ABS) test
   2. T. Pallidum-Hemagglutination Assay (TPHA)
   3. Treponema Pallidum Immobilisation (TPI)

III. CSF Examination for neurosyphilis:

IV. X- ray chest for aortic aneurysm:

E. Treatment:

Stage	Drugs
Primary	- Benzathine penicillin G 2.4 mega units IM once
Secondary	- Benzathine penicillin G 2.4 mega units IM once a week for 2 weeks.
Tertiay (Except Neurosyphilis)	- Benzathine penicillin G 2.4 mega units IM weekly for 3 weeks
Neuro syphilis	- Crystalline penicillin 12 to 24 million units per day IV for 10 days.

If patient is sensitive to Penicillin, Erythromycin is used as an alternative drug.

F. Gonorrhoea Clinical features:

I. In Male:
1. Thick greenish yellow purulent discharge per urethra.
2. Dysuria, increased frequency of urine.

II. In Female:
1. Discharge per vaginum :
2. Dysuria, urgency of urine
3. Vulvo-vaginilis.

III. Opthalmia-neonatorum :

Complications :

In Males:
1. Cystitis
2. Epididymoorchitis
3. Prostatitis
4. Urethral stricture

In Female:
1. Bartholinitis

2. Chronic Urethritis
3. Cervicitis
4. Salpingo-Oophoritis
5. Infertility

Metastatic Complications:

1. Arthritis
2. Conjunctivitis
3. Iritis
4. Endocarditis
5. Meningitis

Investigations:

1. Urethral discharge shows gram negative diplococci.
2. Complement fixation test.
3. Fluroscent antibody test.

H. Treatment:

1. Inj. PPF 8 lacs IM daily for 5 days.
2. Ampicillin or Amoxicillin. 250mg Qid for 5 days.
3. Cetriaxone 250 mg single IM dose plus Doxycycline 100mg orally twice a day for 7 days.
4. Spectinomycin 2gm single IM dose plus Doxycycline 100mg orally twice a day for 7 days.

I. Prevention and Control:

I. Primary prevention:

1. *Health promotion* :
   a. Health education : Regarding,
      i) Safe sexual practices, including the use of condoms.
      ii) Sexual myths.
      iii) Moral values.
      iv) Sex education in schools and colleges.
      v) Avoidance of sexual contact during infective stage.
   b. Legislation:
      i) Control on prostitution. Suppression of Immoral Traffic Act. (SITA).
      ii) Health check up of prostitutes.
2. *Specific protection* : Using condoms.

II. Secondary prevention:

1. *Early diagnosis* : Screening of
   a. High risk groups e.g. Prostitutes
      Labourers.
      Blood donors.
   b. Sexual contacts of known cases (Contact tracing).
   c. Social contacts of known cases (Cluster testing)
2. diagnostic tests as described above.
3. *Prompt and effective treatment* :
   Treatment as outlined above. Follow-up to eliminate carrier states

III. Tertiary prevention:

1. *Diability limitation:* Prompt treatment will prevent the disease from reaching the tertiary stage and thereby limit disability.
2. *Rehabilitation:*
   a. Social
   b. Psychological
   c. Occupational rehabilitation of high risk groups e.g. prostitutes.

13. Typhoid

A. Statistics:

Enteric fever caused by Salmonella has been brought close to elimination in most of the developed countries due to drastic improvement in the water purification, sanitation and food hygiene techniques, in the post World War II era.

In India, as in other developing countries it still contributes significantly to the morbidity. Mortality due to the disease has, however, decreased considerably.

Typhoid is notifiable in Bombay and studies suggest an annual incidence of 0.2 to 0.5 percent, while in India it is estimated to have an incidence of 0.2 to 1 percent.

B. Epidemiology:

I. Agent: Salmonella typhi, Paratyphi

1. Source of infection: Stool and urine of cases and carriers.
2. Mode of transmission : Feco - oral route through
   a. Contaminated water and food.
   b. Direct, through contaminated fingers.
   c. Flies.
   d. Fomites.
3. Period of communicability:
   a. Through out the incubation period and upto 2-3wks.ofthe convalcsence period.
   b. Healthy carriers, both temporary and chronic are known. Chronic carrier stage may last for as long as10-12 yrs. if untreated.
4. Incubation period: 3 days to 3wks.average 10-15days.

II. Host:

Age: Occurs at all ages. More common in young adults.

Sex: More in males than females. Carrier rate is more in females.

III. Environmental and Social factors:

Look for the following points in the history.

1. Poor, water and food hygiene.
2. Overcrowding and low socioeconomic living conditions.
3. Poor sanitation, open air defecation.
4. Prescence of flies.
5. Maximum incidcnce is during rainy season.

C. Clinical features:

1st week presentation:

1. Insiduous Onset - Typhoid.
   Acute Onset- Paratyphoid.
2. Lassitude, headache, bodyache, diarrhoea and abdominal pain.
3. Fever - Intermittent initially, later on continuous (during second week).
4. Bronchitis.
5. Epistaxis.
6. Relative bradycardia (Faget's sign).

2nd week presentation:

1. Apathy.
2. Continuous, high grade fever.
3. Soft, tender splenomegaly.
4. Mild hepatomegaly.
5. Rose spots:
   a. They are common in paratyphi A
   b. They occur between the 7th and the 10th day.
   c. Site–Peri-umbilical (most common) and trunk.
   d. Slightly elevated papules, better seen in fair skinned persons.

3rd week presentation:

1. Toxacmia declines.
2. Patient becomes afebrile.

Complications:

1. Myocarditis.
2. Lobar pneumonia.
3. Bronchitis.
4. Mcningism.
5. Hepatitis.
6. Cholecystitis.
7. Intestinal Perforation (3 to 4%).
8. Intestinal haemorrage (2 to 8%).
9. Orchitis, Pyelitis.
10. Zenker's degeneration of abdominal wall and thigh muscles.
11. Splenic abscess.

D. Investigations:

1. Leukopenia with absence of eosinophils in peripheral smear. (Negeli sign)
2. Blood culture is positive in 80% cases during the 1st week.
3. Clot culture is superior for isolation, and a positive culture may be obtained in less than 24 hours.
4. Marrow culture is the most sensitive method of isolating 'S' typhi.
5. Widal test is done with 'H', 'O' and 'Vi' antigens. Rising 'O' titres in the absence of vaccination is diagnostic. Rise in only 'H' titres can occur in many infections. Rise of 'Vi' titres is suggestive of chronic carrier stage.
6. Stool culture is positive in the 3rd week.
7. Urine culture is positive in the 4th week.

E. Medical Treatment:

1. Diet: Liberal liquid diet during the febrile stage.
2. Bed Rest: Untill one weck after the defervescence stage.

3. Drugs	Dose
a. Chloramphenicol	500mg 6hourly till the temperature become normal, then 500mg 8hourly for 10 days
b. Ampicillin or Amoxicillin	500mg 6hourly for 10 days
c. Co-trimoxazole	2tablets 12 hourly for 10 days or 1 gm IM-12 hourly.
d. Ciprofloxacin	500mg- 750mg twice a day orally or 200mg IV 12 hourly for 10 days.
e. O-floxacin	200mg twice a day
f. Pefloxacin	400mg twice a day.
g. Furazolidone	200mg thrice a day.
h. Cephalosporine	500mg thrice a day.
i. Steroids	They can be used to combat toxaemia and should be avoided during the 3rd week.

F. Prevention and Control:

I. Primary prevention:

1. *Health promotion:* Health education: Regarding, good sanitation, personal hygiene, water and food hygiene, fly control measures. Avoid eating uncovered and unhygeinic food. Concurrent disinfection of infective materials. Legislation: To control unlicensed vendors, prevent the sale of unhygeinic and adulterated food and quality control of eating establishments.

2. *Specific protection:*
   a. Immunoprophylaxis : The vaccine is given in two doses, 6wks. apart, (0.5ml. subcutaneous). Gives 80% protection. Booster doses are required every 3yrs.
   b. Chemoprophylaxis: Is not useful in this disease.

II. Secondary prevention:

1. *Early diagnosis:* By methods discussed above.
2. *Prompt and effective treatment* : As out- lined above. Follow-up for 3months. Diagnosis and treatment of carrier state is equally important.

III. Tertiary prevention: Has no role in this disease.

14. Hepatitis (HAV)

A. Statistics:

Essentially a disease of poor sanitary conditions, this disease has declined to negligible levels in developed countries. It is very much, a public health problem, in developing countries and appears in sporadic, endemic and epidemic forms. Most studies in India suggest that 90-95% of the population has antibodies against HAV.

B. Epidemiology:

I. Agent: Hepatitis A virus

1. *Source of infection* : The infected stool of the symptomatic and asymptomatic cases.
2. *Mode of transmssion :*
   a. Mainly feco-oral route.
   b. Rarely parenteral or sexual.
3. *Period of communicability* : Two weeks before and upto one week after the onset of icterus.
4. *Incubation period* : 15-50days.

II. Host:

1. Age: At all ages, clinically more severe after 20yrs. of age.
2. Sex: Equal in both sexes.

III. Enironmental and Social factors:

1. Poor sanitation, open air defecation.
2. Poor water and food hygiene.
3. Presence of flies.
4. Over crowding and low socioeconomic living conditions.
5. History of eating food outside frequently.

C. Clinical features:

I. Pre-icteric phase : It usually lasts for 4 to 7 days.

1. Anorexia
2. Malaise
3. Fever: It disappears at the onset of jaundice
4. Abdominal pain
5. High coloured urine
6. Mild, soft and tender hepatomegaly

II. Icteric phase:

1. Jaundice
2. Nausea, vomiting
3. Weight loss
4. Stool may be clay coloured.

Complications :

1. Hepatic Encephalopathy
2. Cirrhosis of liver
3. Hepato-renal syndrome.

D. Investigations:

1. Urine for Bile salt and Bile pigment examinations.
2. Liver function tests :
   a. Raised serum aminotransferase enzyme.
   b. Elevated total and conjugated bilirubin.
3. Viral antigen and antibodies.
4. Ultrasonography of liver.

E. Medical treatment:

1. Bed rest till serum enzymes come to normal
2. High protein, High carbohydrate diet.
3. Supportive treatment.
   a. Vitamins
   b. Pancreatic enzymes
   c. Appetite stimulators like cyproheptidine.
4. Treatment of complications.
5. Corticostcroids: It is indicated in
   a. Patients with chronic hepatitis without Australia Antigen.
   b. Prolonged cholestasis with deep jaundice.
6. Ribavarin : It is an anti-viral drug tried in hepatitis A infections.

CHARACTERISTICS OF VARIOUS TYPES OF HEPATITIS

Hepatitis	A	B	C	D	E
a. Incubation Period(days)	15-50	40-180	15-180	28-140	22-60
b. Epidemics	Present	Uncommon	Uncommon	Uncommon	Large epidemics
c. Route of Infection					
1. Faccal excretion	Yes	No	No	No	Yes
2. Faeco-oral	Yes	No	Unknown	Unknown	Yes
3. Percutaneous	Possible but rare	Yes	Yes	Yes	No
d. Sexual contact	No	Yes	Probable	Probable	No
c. Fatal	Usually not	Yes	Unknown	Unknown	No
f. Clinical features:					
1. Course					
i) Chronic Hepatitis	No	5-10%	In50% of cascs	Yes	No
ii) Carrier state	No	Yes	Yes	Yes	No
iii)Risk of development of hepatoma	No	Yes	Probably increased	Variable	No
2. Effect on Pregnancy	yes	No	No	No	Yes High mortality rate in Pregnant women.

F. Prevention and control:

I. Primary prevention:

1. *Health promotion:*
   a. Health education regarding, water and food hygiene, proper sanitary disposal of faeces and other waste. Prevention of fly breeding and fly control measurcs. Prevention of open-air defecation and eating of uncovered, unhygeinic food. Prevention of transmission by infected patient.
   Superchlorination of water, at source, during epidemics. Advise, drinking of water only after boiling for 5 - 10 mins.
   b. Legislation against sale of unhygeinic and adulterated food. Vigorous quality checks of hotels, restaurants and other licensed eating establishments. Prevent the sale of food articles by unlicensed vendors.
2. *Specific protection:*
   a. Immunoprophylaxis:
      i) Vaccine is under trial.
      ii) Human Ig for close contacts of cases particularly those with lowered immune status.
   b. Chemoprophylaxis: Not known.

II. Secondary prevention:

1. *Early diagnosis* :Through methods mentioned above.
2. *Prompt and effective treatment:* As outlined above.
   Follow-up for 3 months after recovery.

III. Tertiary prevention: Has no role in this disease.

15. Poliomyelitis

A. Statistics :

Essentially a disease due to poor sanitation, poor food, hygeine and drinking water contamination, has been, virtually eliminated from the developed countries, but continues to claim many victims, and also leads to deaths in many cases, in the developing countries.

In India

Prevalence (among 0-5yrs. children) - 12per 1000

Incidence (among 0-5yrs. children) - 2.5per 1000

B. Epidemiology :

I. Agent: Polio Virus Types I, II and III, Type I is responsible for epidemics.

1. Source of infection : Stool and oropharyngeal secretions of clinical and subclinical cases (proportion of clinical to subclinical cases is 1:1000 in children and 1:100 in adults)
2. Mode of transmission:
   a. Feco-oral route through contaminated water, food and by contaminated fingers.
   b. Droplet infection, through coughing and sneezing during the acute stage.
3. Period of communicability: All through the incubation period and upto one week after the onset of symptoms. The virus is excreted in the stools for as long as 3months some-times. Chronic carrier states do not exist in this disease.
4. Incubation period: 3-30 days, average 10 days.

II. Host:

1. Age : 80% cases are below 2yrs. of age and the rest below 6 yrs.
2. Sex: Male to Female ratio is 3:1
3. Provocative poliomyelitis is caued by trauma, operative procedures or administering IM injections during the pre paralytic stage.

III. Environmental and Social factors:

1. Maximum incidence is during monsoon.
2. Poor sanitation and open air defecation.
3. Poor water and food hygiene.

C. Clinical features:

I. Prodromal stage:

1. Coryza, cough, sore-throat.
2. Vomiting, diarrhoea.
3. Head-ache, irritability, drowsiness, fever.

II. Pre-paralytic stage: (signs of meningism)

1. Head-ache, vomiting, fever
2. Neck and spinal stiffness.
3. Fasciculation.

III. Paralytic stage :

1. Spinal form.
   a. Flaccid, asymmetrical limb involvement (commonly lower limb)
   b. Areflexia without sensory loss.
2. Bulbar from.
   a. Palatal and pharyngeal muscles paralysis.
   b. Respiratory muscles paralysis.

Clinical variants:

1. Inapparent infection (95%)
2. Abortive (4 to 5%)
3. Non-paralytic (0.5%)
4. Paralytic (0.1 %)

D. Investigations:

1. Mild leukocytosis
2. Neutralising and complement fixing antibodies in serum during 1st and 2nd week.
3. C.S.F. : Pressure and protein-slightly increased.
   Cells, fever, 100-500 cells/mm^3 Initially neutrophilia, later lymphocytic response.

E. Treatment:

For pre-paralytic stage

1. Bed Rest
2. Sedation
3. Fomentation

For Paralytic stage

1. Splint
2. Physiotherapy
3. Prevention of pressure sores and care of sphincter
4. Respirator for respiratory paralysis.

F. Prevention and control:

I. Primary prevention:

1. Health promotion:
   a. Health education regarding, good sanitalion, personal hygiene, water and food hygiene, avoid eating uncovered and unhygeinic if food, concurrent disinfection of infective materials.
   b. Legislation: To control unlicensed vendors, prevent the sale of unhygienic and adulterated food, and quality check of eating establishments.
2. Specific protection:
   a. Immunoprophylaxis:
      i) Live, Oral polio (Sabin) vaccine is given at 6,10,14 weeks *of* age and one booster at 18months as per the UIP schedule. (Additional zero dose at birth, is now advocated). This vaccine is said to give life long immunity.
      ii) Inactivated, Injectable (Salk) vaccine given in 3 doses, 4-6 wks. apart, followed by a booster after 1yr. is equally useful.
   b. Chemoprophylaxis: does not exist for this disease.

II. Secondary prevention:

1. Early diagnosis: As mentioned above and by active and passive surveillance.
2. Prompt and effective treatment : By steps outlined above.
3. Ring immunization and mop up survey and immunization of the area where the case has occurred is advocated.

III. Tertiary prevention:

1. Physical rehabilitation:
   a. Physiotherapy.
   b. Reconstructive surgery.
   c. Prosthesis and physical aids like crutches and special shoes.
2. Occupational rehabilitation: Occupational therapy.

16. Cholera

A. Statistics :

Cholera is a disease that has been known since ages. lt occurs as endemic, sporadic, epidemic and pandemic. Being a disease of poor sanitation, poor water and food hygeine it has been almost eliminated from the developed countries.

Cholera is notifiable all over India as well as to WHO. Mortality due to cholera has decreased drastically in India, and the incidence of the disease has also decreased significantly in the last decade.

B. Epidemiology :

I. Agent: Vibrio cholerae

1. Source of Infection: Stool and vomitus of cases and carriers of cholera.
2. Mode of transmission: Feco-oral route.
   a. Through contaminated water, and food.
   b. Through fomites. .
   c. Direct contact by contaminated fingers.
   d. Flies.
   Ask for the presence of cases in the family and close contacts.
3. Period of communicability:
   a. Through out the period of incubation and till the period of convalescence (2- 3wks.)
   b. Healthy contact carriers, with subclinical infection, upto 10 days.
   c. Chronic carrier state upto 10yrs sometimes, if untreated.
4. Incubation Period: 6 hrs. to 6 days.

II. Host :

1. Age: All ages, more in children.
2. Sex: Both sexes equally affected.

III. Environmental and Social factors :

Look for the following points in the patient's history.

1. Poor, water and food hygiene.
2. Poor sanitation, open air defecation.
3. Presence of flies.
4. Over crowding and low socioeconomic liv ing conditions.
5. Prescence of large numbers of mobile and migrant persons, associated with poor water and food hygiene, and poor facilities for waste disposal eg. during melas, fairs and yatras.

C. Clinical features :

I. Stage of evacuation:

1. Profuse vomiting.
2. Frequent loose motions, watery, copious, with flakes of mucus (Rice water stool)

II. Stage of collapse :

1. Hypovolaemic shock, due to massive di arrhoea and vomiting.
2. Tachycardia and Tachypnoea.
3. Oliguria.
4. Cold and clammy skin.

III. Stage of recovery :

1. Vomiting and loose motions decrease.
2. Hydration improves.
3. Normal temperature returns.

Atypical presentation.

a. Cholera Sicca :
   It is a fatal variety, in which there is very little or no diarrhoea or vomiting and patient develops collapse very rapidly with overt manifestations.

Complications :

1. Hypovolemic shock.
2. Acute renal failure.
3. Electrolyte disturbances.
4. Enteritis.
5. Cholecystitis.
6. Stroke in the elderly patient.

D. Investigations :

1. Leukocytosis with polymorphonuclear predominance.
2. Stool swab, for demonstrating darting motility, of V. cholerae.
3. Stool culture for V. cholerae.
4. Slide agglutinalion using polyvalent cholera diagnostic serum.

E. Medical Treatment :

I. Correction of fluid and electrolyte imbalance.

1. Initially, venous intra infusion like Saline, Haemacele.
2. Once vomiting subsides oral rehydration should begiven.

II. Drugs :

1. Tetracycline : 500 mg 6-8 hourly.
   OR
   Doxycycline 300 mg daily
2. Chloramphenicol 500 mg 6 or 8 hourly
3. Furazolidone : 200 mg twice a day.

F. Prevention and Control

I. Primary prevention :

1. *Health promotion* :
   a. Health education regarding :
      i) Good sanitation, personal hygiene.
      ii) Water and food hygiene.
      iii) Fly control measures.
      iv) Avoid eating uncovered and unhygienic food.
      v) Steps to prevent spread of infection from known cases, e.g. disinfection of infective materials.
      vi) Legislation against thc sale of unhygienic and adulterated food. Quality checks on eating establishments. Prevent unlicensed vendors from selling prepared food items.

   b. Specific protection :
      i) Immunoprophylaxis: Cholera vaccine is of no value in the prevention and control of cholera.
      ii) Chemoprophylaxis : Single oral dose of doxycyline (300mg) and 6mg/ kg for children. Tetracyline for 3 days may be given to close contacts. However it has no use as a mass prophylaxis method.

II. Secondary Prevention:

1. Early diagnosis: Through methods mentioned above.
2. Prompt and effective treatment : As outlined above. Follow-up for 3 months. Diagnosis and treatment of carrier state is equally important.

III. Tertiary prevention : Has no role in this disease.

17. Diphtheria

A. Statistics:

Diphtheria has been virtually eliminated from most of the developed countries.lt has declined considerably in the developing countries over the last two decades. Recent studies in India suggest an annual incidence rate of about 5-8 per million population.

B. Epidemiology:

I. Agent : Corynebacterium diphtheriae

1. Source of infection : Cases and carriers. Secretions from their nose, throat and skin lesions.
2. Mode of transmission :
   a. Droplet infection and droplet nuclci through air.
   b. Direct contact with skin lesions.
   c. Fomites (less commonly).
3. Period of communicability:
   a. About a month after the onset of signs and symptoms and a week before that.
   b. Carrier states are known to occur for 3-4 months or longer, unless adequately treated.
4. Incubation period : 2-7 days.

II. Host :

1. Age : Maximum incidence during 6 months to 6 yrs. Can also occur at later ages in susceptible hosts.
2. Sex : Equal in both sexes.

III. Environmental and Social factors :

1. Cases occur throughout the year.
2. Over crowding and poor ventilation. .
3. Lower socio-economic groups.

C. Clinical features :

1. Nasal diphtheria :

a. Unilateral or bilateral nasal discharge, serous initially, often blood stained later on. A thick membrane may be visible on the mucosa of the anterior part of nasal septum.

2. Pharyngeal diphtheria :

Presence of membrane over tonsils and or pillars of fauces. The membrane is thin and glistening white in the early stages. It is adherent, hence bleeds on forcible removal. Patient with severe disease (malignant diphtheria) may have oedema around submandibular areas and the anterior neck giving a charactcristics "bullneck appearance".

3. Laryngeal diphtheria : More common in infants.

a. Hoarsness of voice.
b. Brassy cough.
c. Noisy breathing.

Complications :

1. Toxemia :a. Fever
   b. Pallor
   c. Listlessness
   d. Tachycardia
   e. Weakness
2. Cardiovascular system involvement:
   a. Myocarditis
   b. Arrythmias
   c. Peripheral shock
3. Nervous system involvement:
   a. Peripheral neuritis
   b. Cranial nerves dysfunction
   c. Cerebral infarction (rarely)

D. Investigations:

1. CBC shows marked neutrophilic leukocytosis with high ESR.
2. Demonstration of the organism in
   a. Smear
   b. Culture

E. Medical treatment:

1. Complete bed test
2. Liquid diet
3. Local antiseptic gargles
4. Antitoxin:
   Diphtheria antitoxin (DAT) is given in the dose of 50,000 units to 100,000 units Intravenous, after test a dose.

5. Antibiotics:
   Injection Crystalline penicillin 20 lacs units IV 6 hourly for 10 days.
6. Management of complications like
   a. Laryngeal involvement
      - Back rest
      - Oxygen
      - Tracheostomy SOS
   b. Shock - IV fuids
      - Ionotropic support
   c. Respiratory muscle paralysis
      - Ventilatory support

F. Prevention and Control :

I. Primary Prevention :

1. Health promotion: Health education relatedto overcrowding, ventilation and the need to take DPT and DT vaccines for children below 6 yrs of age.
2. Specific protection: Diphtheria toxoids given along with pertussis and tetanus.
   a. Immunoprophylaxis : DPT, and DT are part of the UIP. Should be administered to all children below 6 yrs of age as per the UIP schedule.
   b. Chemoprophylaxis: Erythromycin, Ampicillin.

II. Secondary prevention:

1. Early diagonosis
   a. Screening of close contacts by taking throat swabs for smear
2. Prompt and effective treatment
   a. As outlined above.
   b. Isolation of cases.
   c. Concurrent disinfection of infective material.
   d. Follow up, for avoiding carrier states

III. Tertiary prevention :

1. Disability limitation: Treatment at early stages prevents the heart and peripheral nerves from being affected.
2. Rehabilitation: Is not required.

18. Diarrhoea and Dysentery

A. Statistics:

As per National Diarrhoeal Diseases Control Programme, diarrhoea is responsible for 20-30% of death of children below 5 yrs and 10% of deaths below 1 yrs. It is estimated that 1.5million children below 5 yrs of age suffer from acute diarrhoea every year in Asia, Africa and Latin America.

Being a disease of poor sanitation it is virtually eliminated from the developed countries.

B. Epidemiology:

I. Agent:

1. Source of infection : Fecal matter of cases and carriers, human and animal reservoirs.
2. Mode of transmission : Feco-oral route;
   a. Water and food contamination with faeces.
   b. Contaminated fingers.
   c. Transmission by flies.
3. Period of communicability : Till such time as the organism is excreted in the faeces.
4. Incubation period : Varies, depending on the causative organism.

II. Host:

1. Age : More in young children and the aged.
2. Sex: Equal in both sexes.
3. Malnutrition predisposes to diarrhoea and diarrhoea further worsens malnutrition, propagating the vicious cycle of infection and malnutrition .

III. Environmental and Social factors:

1. Poor water and food hygiene.
2. Poor personal hygiene.
3. Overcrowding and poor sanitation.
4. Fly breeding.
5. Poor facilities for waste disposal.
6. More in late summer and during monsoon.

C. Clinical features:

1. Loose motion: Initially watery (diarrhoea) later on with mucus and/or blood (dysentery).
2. Abdominal pain.
3. Vomiting: Due to severe abdominal pain which stimulates the vagus nerve.
4. Fever :Usually not present, but may be of low grade.

D. Investigations:

1. CBC with ESR
   Leukocytosis with band forms in the peripheral smear.
2. Stool examination: It may reveal.
   a. Trophozoites indicating Amoebiasis.
   b. Vibrio Cholerae is usually seen in hanging drop preparation.
   c. Giardia lambia.
3. Stool culture for shigellosis.

E. Medical Treatment:

1. Correction of dehydration.
2. Elcetrolyte balance.
3. Chemotherapy
   a. Ampicillin, Co-trimoxazole for Bacillary dysentery.
   b. Tetracycline for V. cholerae.
   c. Metranidazole / Tinidazole for Giardiasis / Amoebiasis.
4. Pectin-Kaolin binder may be useful.
5. Anti-motility drug like lomotil or loperamide should be avoided.

F. Prevention and Control :

I. Primary prevention:

I. Health promotion:
   a. Health education about personal hygiene, sanitation and waste disposal, prevention of fly breeding, food and water hygiene. Avoidance of eating uncovered and unhygienic food from road side vendors.
   b. Legislation:
      i) For quality check on hotels and eatiling establishments.
      ii) Licensing and Certification of vendors and food suppliers.
      iii) Health check up or food handlers.
2. Specific protection :
   a. Immunoprophylaxis: Immunizalion is available against Salmonella, Shigella E. coli and certain viruses, but not yet used for mass prophylaxis in our country.

II. Secondary Prevention:

1. Early diagnosis : Using the tests described above.
2. Prompt and effective treatment : As outlintd above and follow-up to avoid carrier stutes.

III. Tertiary Prevention: Has no role in this disease.

19. Alcoholism

A. Definition :

A psychological condition where alcohol is required for adequate functioning and the individual continues drinking despite social and/or occupational problems.

Alcohol dependence is a state, where alcohol consumption increases progressively and physical signs appear on withdrawal of alcohol from the individual.

B. Epidemiology:

Social, Cultural and Demographic factors:

1. Poor socio-economic conditions.
2. Poor education.
3. Industrialization and rapid urbanization.
4. Family and peer influences.
5. Religious and cultural influences.
6. Cheap and easy availability.
   It is the cheapest source of escapism for an average labour class person.
7. Poor legislative control and poor enforcement of existing legislations.

C. Clinical features :

1. Due to alcoholism
   a. Acute psychotic behaviour
   b. Alcoholic hallucinations
   c. Paronia
2. Alcohol Withdrawal Syndrome (Delirium tremens)

After excessive consumption followed by abstinence for 24 hrs patient develops Delirium Tremens.

   a. Sleeplessness
   b. Restlessness
   c. Loss of appetite
   d. Weakness.
   e. Tremors.
   f. Hallucinations and delusions.
   g. Convulsions can occur.

3. Wernicke's Encephalopathy :
   It occurs due to thiamine deficiency and is characterised by:
   a. Mental disturbances.
   b. Paralysis of eye movements.
   c. Ataxia of gait.
4. Korsakoff's psychosis: It is characterised by:
   a. Disorientation.
   b. Amnesia for recent events.
   c. Confabulation.
   d. Euphoria.

D. Complications:

1. Alcoholic hypoglycemia.
2. Gastritis.
3. Alcoholic liver disease.
4. Polyneuropathy.
5. Beri-beri.
6. Pellagra.
7. Amblyopia.
8. Sub-acute combined degeneration of the spinal cord (vitamin B12 deficiency).
9. Alcoholic Cerebellar degeneration.
10. Dementia.
11. Vitamin E deficiency characteried by dysarthria, ataxia and polyneuropathy.
12. More prone to develop tuberculosis.

E. Investigations:

1. HB, CBC and ESR. Patient may megaloblastic anaemia.
2. Liver function tests :
   a. Gamma Glutamyl transpeptidase (GGTP). It is the first enzyme to rise in alcoholic patients.
   b. Albumin: Low albumin suggests chronic liver damage.
   c. SGOT / SGPT : Raised. Ratio of 2:1 or more is found in case of alcoholic hepatitis.
   d. Bilirubin increases in severe alcoholic liver damage.
3. Stool: May show presence of blood as in the case of:
   a. Gastritis.
   b. Bleeding varices.
4. Ultra-sonography of abdomen.
5. Isotope liver scan, to diagnose cirrhosis of liver.
6. X-ray chest to rule out Pulmonary Tuberculosis.

7. ECG:- It is useful in the diagnosis of :
   a. Alcoholic Cardiomyopathy.
   b. Arrythmias.

F. Medical treatment:

Conditions	Treatment
1. Hypoglycemia	Intra-venous concentrated glucose solution
2. Wernicke's Encephalo-pathy	Injection Thiamine l00mg lm
3. Pellagra	Nicotinic acid (25mg) 50mg thrice a day
4. Withdrawal Syndrome	a. Sedation : Diazepam or chlordiazepoxide 5 to 15mg thrice a day. b. Plenty of Glucose. c.Injection Thiamine.
5. Korsakoff's Psychosis	a. Sedation: Diazepam or Chlordizepoxide b. Anti-Psychotic drugs like, Haloperidol, Chlorpromazine.
6. Sub-acute combined degeneration of the spinal cord.	Injection Vit-B_{12}100μgm Im daily for 10 days then 1000μgm once a month.

Cirrhosis of liver

G. Clinical features:

1. Asymptomatic - Initial stage.
2. Signs of liver cell failure.
   a. Generalised weakness.
   b. Icterus.
   c. Foetor hepaticus
      Smell in the breath of a person with liver cell failure akin to a freshly opened corpse or a dead mouse.
   d. Bleeding diathesis.
   e. Flapping tremor.
   f. Ascites.
   g. Endocrine manifestations:
      i) Gynaecomastia.
      ii) Loss of secondary hair.
      iii) Loss of libido.
      iv) Palmar erythema.
      v) Testicular atrophy.
      vi) Spider naevi.
3. Signs of Portal hypertension:
   a. Splenomegaly.
   b. Evidence of varices.
   c. Dilated veins.

H. Complications:

1. Liver cell failure.
2. Portal hypertension and its sequelae.
3. Hepatoma.
4. Spontaneous bacterial peritonitis.
5. Hepatorenal syndrome.
6. Hypersplenism.
7. Coagulopathy.

I. Investigations:

1. Anaemia: - Normochronic and Normocytic. Hypochromic if variceal blood loss occurs.
2. Liver function tests.
   - Hypoalbuminemia.
   - Normal Serum enzymes.
   - Prolonged Prothrombin time.
3. Ultrasonography:
   - Hyper-echoic liver.
   - Splenomegaly.
   - Enlarged Portal vein.
4. Upper G I Scopy } To demonstrate varices
5. Barium swallow }
6. Radio isotope liver scan.
7. Liver biopsy.

J. Medical Treatment:

1. High protein and low salt diet
2. Diuretics preferably Anti-aldosterone drug like Spironolactone Amiloride. If required, frusemide can be added.
3. Ascitic tapping
4. Treatment of variceal bleeding.
5. Treatment of other complications.

K. Prevention and Control:

I. Primary Prevention:

1. Health promotion: :
   a. Health education about the physical and social aspects of alcohol addiction, particularly to the high risk population (ie. the labour class)
   b. Legislative control over alcohol production, sale and consumption.

II. Secondary Prevention:

1. Early diagnosis:
   a. Surveys of high risk population.
   b. Surveillance of high risk population.
2. Prompt medical treatment as required.

III. Tertiary Prevention:

1. Physical rehabilitation.
2. Occupational rehabilitation.
3. Psychological rehabilitation.
4. Social rehabilitation.

20. Mental Retardation

A. Definition :

Mental Retardation is defined as below average intellectual functioning capacity, associated with impairment of adaptive functioning.

B. Statistics:

Mental retardation is a world wide problem. About 50% of the total cases are found in the developing countries and about 30% of the total cases are below 15 yrs of age.

In India 1-2% of the population is said to be affected.

C. Epidemiology:

Causes:

1. Idiopathic
2. Pre-natal causes
   a. Chromosomal anomalies.
      i) Down's Syndrome.
      ii) Turner's Syndrome.
   b. Inborn errors of metabolism.
      i) Phenylketonuria
      ii) Galactosemia
      iii) Mucopoly saccharidosis.
   c. Cranial malformations.
      i) Microcephaly.
      ii) Hydrocephalus.
3. Perinatal causes:
   a. Infections. e.g. TORCH.
   b. Physical causes.
      i) Birth trauma.
      ii) Radiation.
   c. Pre-maturity
   d. Intoxications with Bilirubin (Kernicterus)
4. Post natal causes:
   a. Infections
      i) Meningitis
      ii) Encephalitis.
   b. Head injury.
   c. Malnutrition.
5. Endocrine disorders e.g. Cretinism.

D. Clinical Features:

1. Look for family history of mental illness.
2. Anatomical defects e.g.
   Microcephaly, Hydrocephalus
   Malformations of the eyes, ears and nose.
3. Delay in mental and physical milestones.
4. Articulation defects.
5. Scholastic backwardness.
6. Poor general and practical knowledge.
7. Poor IQ. (Intelligence Quotient)

$$IQ = \frac{\text{Mental age}}{\text{Chronological age}} \times 100$$

IQ		
75 - 90	-	Mentally subnormal.
50 - 75	-	Mild mental retardation
25 - 50	-	Moderate " "
below 25	-	Severe " "

E. Investigations:

1. Chromosomal studies.
2. Maternal antibody titers for TORCH antibodies.
3. Biochemical studies.
4. Endocrine studies.
5. EEG and CT Scan.
6. IQ and other studies for asscssment of mental development.

F. Treatment:

No drugs have been found useful. Among some of the drugs tried recently are :

a. Piracetam
b. Ginseng

Many herbal remedies and nature cure therapies are tried, with little benefit.

Even well educated parents submit themselves to religious cures and faith healers, against medical advice.

The treatment is mainly tertiary prevention.

G. Prevention and Control:

I. Primary Prevention:

1. Health promotion:
   a. Health education regarding the causes and how to avoid some of them.

b. Prospective genetic counselling.
c. Marriage counselling.

2. Specific prevention: Good quality MCH care, mainly:
   a. Antenatal care.
   b. Intranatal care.
   c. Postnatal care.
   d. Immunisation and Growth monitoring.
   e. Nutritional supplements.

II. Secondary Prevention:

1. Early diagnosis: By means of special education of:
   a. Medical personnel.
   b. Parents.
   c. Teachers.

   to recognise early signs.

III. Tertiary Prevention:

1. Disability limitation and,
2. Rehabilitation:
   a. Specialised institutions to impart care and training for the mentally handicapped.
   b. Occupational therapy.
   c. Half-way homes and family service programmes.
   d. Public education.
   e. Sterilization of those with moderate and severe Mental Retardation.

21. Malnutrition

A. Statistics:

Malnutrilion is a major problem in all developing countries. Childhood as well as Adult Malnutrition is seen all over Africa, Asia and some parts of Latin America and Europe.

In India the major nutritional problems are

Nutritional Problem	Prevalence
a. P .E.M.	By Clinical examination3%. By Anthropometric 4% in (0-6 yrs. children).
b. Vit A dcficiency	Conjunctival xerosis 5-10%, corneal involvement 0.12% (in 0- 6 yrs. children).
c. Anaemia	By Hb. estimation 90% of pregnant women, and 60% of 0-6 yrs children.
d. Endemic Goitre	Thyroid enlargement in 33% of the community at risk.

B. Epidemiology:

I. Cultural and Social factors:

1. Harmful cultural patterns and habits related to:
   Breast feeding.
   Weaning.
   Feeding of ill children.
   Food taboos.
   Feeding of pregnant and lactating women.
   Repeated pregnancies, lack of spacing.
2. Infections:
   Repeated infections mainly:
   G.I. infections.
   Worm infestations.
   Respiratory infections.
   Measles.

II. Ecological factors:

Factors relating to:

a. Agricultural growth, production, transportation, storage, distribution and research.
b. Poultry farming.
c. Animal husbandry.
d. Pisiculture.
e. Import and export of food materials.
f. Climatic conditions e.g. drought, famines, floods etc.

C. Clinical features:

PEM.

C/F	Marasmus	Kwashiorkor
a. Edema	—	+
b. Wasting	+++	—
c. Growth retardation	++	+
d. Mental changes	Cranky	Apathy.
c. Appctite	++ (present)	absent.
f. Diarrhoea	++	++
g. Skin changes,	+ dry and cracked skin and hypopigmentation	+++ Crazy pavement dermatitis.
h. Hair, hypopigmentcd brownish breaks easily.	+ —	+++ flag sign.
i. Moon face	—	+
j. Enlarged abdomen (liver ++)	—	+

C/F of vit A deficiency.

1. Night blindness (Nyctalopia)
2. Conjunctival xerosis.
3. Bitot's spots.
4. Corneal xerosis.
5. Corneal ulcers.
6. Corneal scars.
7. Keratomalacia.
8. Phrynoderma. (toad skin)

C/F of B plex. deficiency

1. Cheilosis
2. Stomatitis (angular Stomatitis)
3. Apthous ulcers.
4. Swelling and redness of tongue.
5. Nasolabial dysbaecia.
6. Peripheral neuritis.

C/F of Anemia

1. Pale look

2. Pale conjunctiva
3. Kolionychia
4. Pale tongue

Assessment of nutritional status is done on the basis of :

1. Clinical examination
2. Anthropometric examination: - Height, weight, skin fold thickness, Mid-arm circumference.
3. Lab investigations - Hb, Ferritin, Calcium, Iodine, Retinol in the blood and Bioassays' for vitamins.
4. Diet survey.

D. CLASSIFICATIONS OF MALNUTRITION

I. Wellome

Weight / Age	edema	
60 - 80%	- ve	undernourished.
< 60%	- ve	marasmus.
60 - 80%	+ ve	kwashiorkor.
< 60%	+ ve	marasmic kwashiorkor

II. Waterlou

Weight / Age	Height / Age	Weight / Height	Status of malnutrition
⇓	N	⇓	current
⇓	⇓	⇓	past
⇓	⇓	N	chronic

N = normal ⇓ =decreased.

III. Gomez

Weight / Age.	
> 90%	Normal
70 - 90%	Grade I
60 - 75%	Grade II
< 60%	Grade III

IV. I.A.P.

Weight/Age.	
> 80%	Normal
70 - 80%	Grade I
60 - 70%	Grade II
50 - 60%	Grade III
< 50%	Grade IV

E. Treatment of malnutrition

1. Treatment of infections and infestations.
2. Correction of electrolyte imbalance.
3. Correction of hypoglycemia.
4. Correction of hypothermia.
5. Correction of hypocalcemia.
6. Diet : Protein : 3 - 4 gm / kg / day.
   Calories : 170 - 200/ kg / day.
7. Vitamin and mineral supplements.
8. Nutritional rehabilitation if required.

F. Prevention and Control

I. Primary Prevention:

1. Health promotion:
   a. Good ANC, INC, PNC, and promotion of breast feeding.
   b. Low cost weaning foods.
   c. Nutrition education - correct feeding practices.
   d. Family welfare measures.
2. Specific protection
   a. Food fortification with vitamins, minerals, proteins, energy etc.
   b. Food supplements - Vit. A drops, Iron, folic acid.
   c. Immunization of children.

II. Secondary prevention:

1. Early diagnosis:
   a. Periodic surveys.
   b. Regular growth charting (nutritional surveillance)
2. Prompt treatment:
   a. Treatment of infections.
   b. Deworming.
   c. Feeding programmes.

III. Tertiary prevention

1. Nutritional rehabilitation.

Immunization or Growth monitoring case.

Children attending Growth monitoring or Under Five's clinics or immunisation clinics are sometimes kept as cases.

The student is expected to examine the child in the routine way including

Personal History
Family History
Social History and
Environmental History

Clinical examination

The student should highlight any discrepancies in the Growth and Development card or immunisation card.

The viva is usually on the following topics.

1. Immunisation schedules.
2. Cold chain.
3. Individual vaccines.

4. Vaccine contraindications and Sterilization of needles and syringes.
5. National,Immunisation goals and family welfare goals by 2000 AD.
6. Malnutrition, Growth Monitoring etc.

MTP Case

A case for MTP is sometimes kept. The discussion and viva is usually on the following topics.

1. Antenatal care.
2. MTP (provisions of the act).
3. Adoption and Foster parents (in case of unmarried women, over 20 wks of gestation)
4. Psychosocial problems related to MTP and unmarried pregnancy.
5. Contraceptive devices.
6. Sex education in school and colleges.
7. National family welfare goals by 2000 AD.

The student is expected to examine the case as a case of pregnancy, and is suppose to enquire into the social and environmental history.

22. Antenatal Case

INTRODUCTION : Maternal mortality rate in India is still unacceptably high as compared to many other countries, according to the SRS (1991) estimated M.M.R. is 62.9 per 100,000 women in the reproductive age group of 15 to 44 years or 3.4 per 1000 live births. The safe motherhood programme aims at reducing the maternal mortality rate to less than 2 per thousand live births by the year 2000.

Maternal death is defined as the death of a women during pregnancy or within 42 days of the termination of pregnancy, irrespective of the duration or site of pregnancy, from any cause related to or aggravated by pregnancy or its management, but not from any accidental cause.

Major causes of maternal deaths in India, are:

a)	Anemia -	20%
b)	Hemorrhage	20%
c)	Sepsis	13%
d)	Obstructed Labour	12%
e)	Abortions	11 %
f)	Eclampsia	09%
g)	others	15%

A large proportion of these deaths can be averted by providing essential maternal care to all pregnant women.

Antenatal Care is defined as the care that is provided to the pregnant women with the aim of ensuring material and fetal well being, with following specific objectives :

a) To reduce maternal, perinatal and infant mortality and morbidity.
b) To monitor the progress of pregnancy.
c) To identify maternal and fetal complications and refer or treat such cases in a timely and appropriate way.
d) To educate mothers regarding diet, rest, personal hygiene, warning signs of pregnancy, and to prepare them for labour.
e) To educate the mothers on aspects of child health such as breast feeding, weaning, immunization, growth monitoring.
f) To encourage the mother to follow up for post natal and under - 5 clinic.
g) To sensitize the mother for the adoption of a small family norms.

COMPONENTS OF ANTENATAL CARE :

ANTENATAL HISTORY TAKING : A detailed personal, socioeconomic, family, medical and obstetric history must be taken. Some of the important questions to be asked are:

a) LMP: It is important to note the LMP to calculate the EDD and to correlate the progress of pregnancy as assessed clinically with that calculated from the LMP.
b) Age of the mother: Age less than 18 years and over 30 years is considered as a high risk
c) Order of Pregnancy: Primi and grand multiparous women are at a higher risk of complications.
d) Birth interval: Birth Interval of less than two years is a important risk factor.
e) Problems during previous pregnancy: Eclampsia, APH, abortions, still births, premature births, malpresentation, prolonged labour, PPH, LSCS.
f) Systemic illness in the past. : Heart disease, diabetes, hypertension, tuberculosis, epilepsy are important illnesses.
g) Dietary history: A detailed dietary history should be obtained. From the diet, the daily calorie and protein intake must be calculated and its adequacy ascertained. Pregnant women require an additional 300 Kcal and 15 gm of additional protein per day throughout the pregnancy.
h) Present complaints: Breathlessness, palpitations, excessive tiredness, headaches, blurring of vision, edema feet/hands/face, fever, bleeding/leaking PV, abdominal pain, any other complaints.

ANTENATAL CLINICAL EXAMINATION: A thorough general, systemic and per abdominal examination should be performed, at each visit. A PV examination is performed only once during the first trimester to confirm pregnancy, assess the duration of pregnancy and to rule out any pelvic abnormalities such as ovarian cysts/tumors etc.

Unless it is otherwise indicated (e.g. incompetent os), the next PV examination is generally performed after 36-37 weeks of gestation.

GENERAL EXAMINATION: During each visit the weight and BP are recorded. Pallor and edema are looked for:

a) Height: Height less than 145 cm is a risk factor.

b) Weight: Weight gain is monitored at each visit. On an average, mothers gain 10 to 12 Kg of weight throughout the pregnancy. A weight gain of more than 3 kg in a month should arouse suspicion of PIH. A poor weight gain is indicative of LBW/IUGR.

c) Blood Pressure: B.P. is recorded at each visit. A B.P. of greater than 145/90 mm Hg on two consecutive readings taken at least four hours apart, which develops after 20 weeks of gestation is suggestive of pre-eclampsia.

d) Pallor: Nails, tongue, palms and palpebral conjunctiva are examined for pallor.

e) Edema feet/hands/face: Often during late pregnancy, pitting ankle edema is seen, which does not warrant any special attention. If however, the edema feet/hands/face is accompanied by albuminuria and high B.P, pre-eclampsia should be suspected.

f) Thyroid swelling, oral and dental hygiene, varicose veins/piles, enlarged lymph nodes should be examined.

g) Breast Examination: Breast must be examined in all cases. Darkening of the primary areola, formation of secondary areola, Montgomery's tubercles are the changes seen in pregnancy. Nipples must be examined to rule out flattened/retracted nipples. If present, the women is asked to gently manipulate the nipples between the index finger and the thumb throughout the course of the pregnancy.

SYSTEMIC EXAMINATION: A detailed systemic examination is performed to rule out any underlying diseases e.g. heart disease, or respiratory diseases.

ABDOMINAL EXAMINATION:

INSPECTION: The size and shape of the abdomen is noted. The Linea Nigra/ Striae Gravidarum or Linea Albicantes may be seen. Scars of previous surgery may be present (e.g. LSCS).

PALPATION : Assess the fundal height.

1. 12 weeks - Just palpable above the symphysis pubis.
2. 16 weeks - upto lower 1/3rd of the distance between symphysis pubis and umbilicus.
3. 20 weeks - upto 2/3rd of the distance between symphysis pubis and umbilicus.
4. 24 weeks - at the level of the umbilicus.
5. 28 weeks - upto lower 1/3rd of the distance between umbilicus and xiphisternum.
6. 32 weeks - upto 2/3rd of the distance between umbilicus and xiphisternum.
7. 36 weeks - at the level of the xiphisternum.
8. 40 weeks - fundus descends to the level of 32 week, as the head engages, but the flanks feel fuller.

Palpate the fundus (using fundal grip), the small parts and back of the fetus (using lateral grip) and assess the presentation, altitude and engagement using the pelvic grip. Palpation also reveals whether the uterus is relaxed and the Liquor amnii is adequate.

AUSCULTATION:
Fetal heart sounds are auscultated for an entire minute using a fetoscope or stethoscope. Normal fetal heart rate is 140 per minute. A fetal heart rate of less than 120 per minute or greater than 160 per minute is suggestive of fetal distress.

ANTENATAL INVESTIGATIONS : Basic investigations which must be performed are:

1. Hemoglobin - Hb less than 10 gram percent is defined as anemia.
2. Blood grouping and Rh typing.
3. Urine - for albumin and sugar.
4. VDRL.

If indicated and available, then other investigations may be performed such as:

1. Urine - routine and microscopy.
2. Stool - routine.
3. Australia Antigen.

4. ELISA for HIV.
5. USG of abdomen & pelvis
6. FBS/PLBS.

ANTENATAL HEALTH EDUCATION : Mothers are advised on the following topics:

Diet : Mothers are told to increase the daily intake of their normal diet. There are many false beliefs regarding food items which should and which should not be eaten during pregnancy e.g. hot foods like papaya, egg, chicken should not be consumed. Such misconceptions should be cleared and a women should be informed about the nutritive value of the locally available and cheap food items. Simple recipes of their preparations should be given to them. The importance of increased calories, protein, iron and calcium intake and its consequences on the mother and the fetus should be explained. Mothers are encouraged to take iron and folic acid tablets regularly.

Physical work, rest and travel : The mothers must get atleast eight hours sleep at night 'and two hours in the afternoon. Hard manual labour and lifting of heavy objects must be avoided. Short periods of rest between work is advised, Long tiresome journeys are to be avoided during the first and the last trimester.

Tobacco, alcohol and drugs : Tobacco and Alcohol should be strictly avoided. All drugs are best avoided during pregnancy and the mothers are to be warned against any form of self medication. Only drugs prescribed by the treating physician/ obstetrician should be taken.

Personal hygiene : Daily bath and change of clothes should be encouraged.

Warning signs of pregnancy : Mothers are advised about the danger signs of pregnancy such as leaking/bleeding PV, severe abdominal pain, decreased or absent fetal movements, blurring of vision etc. and are advised to seek medical attention immediately.

Preparing for labour : Mothers are told about the symptoms and signs of early labour. Mothers are advised to get admitted at the health facility when these symptoms appear. If they choose to have the delivery at home. Necessary preparations should be made. Mothers are now being provided with a delivery kit similar to the one provided to the TBA. If this is not available with the mother, a new blade should be kept ready. The thread for timeing the umbilical cord of the fetus should be boiled for 20 minutes and dried in the sun. Pieces of cotton cloth should be washed and dried in the sun. This will be needed for receiving the baby, as well as tor conducting delivery. The room where the delivery is to be conducted should be cleaned and well lit up. Relatives of the mother should contact the trained Dais in their village at the on set of labour. All women should be told about the nearby health institutions where they can be taken in case of any complications. The family and neighbours must make arrangements for transport well in advance so that timely transfer of the mother is possible in case of emergency.

CHILD HEALTH ISSUES

1. Newborns should be put to the breast immediately after birth. Newborns should be given colostrum. No pre-lacteal feeds should be given and exclusive breast feeding should be carried out for four to six months.
2. Newborns should be received in a clean cloth. No bath must be given immediately as this can lead to hypothermia and vigorous scrubbing of the vernix leads to damage of the skin, causing infections.
3. BCG and DPV should be given before discharging from the hospital. The immunization schedule and its importance should be explained.
4. Birth weight must be recorded soon after birth and also in case of home deliveries within two days The importance of the birth weight in relationship to the health of the child and subsequent growth and development of the child should be explained.
5. Mothers are explained about the need and the procedures for the registration of the birth.

FAMILY PLANNING METHODS:

The need for family planning and the various available methods should be explained. At this stage, the woman has just undergone the hardships of pregnancy and labour, is very receptive to the suggestion of adopting contraceptive methods. This has been utilized in the All India Hospital Post Partum programme 1979.

SAFE MOTHERHOOD STRATEGIES AND INTERVENTIONS :

The aim of the safe motherhood programme is to reduce the maternal mortality rate to less than 2 per thousand deliveries by the year 2000 per AD To achieve this, the programme the following strategies and interventions are devised.

1. **Early registration:** It is customary in many parts of our country for women to register in the seventh month of pregnancy. Many untoward complications such as abortions, APH can be averted if the mothers register earlier in the pregnancy. It is recommended that women should register for ANC as soon as pregnancy is known to them. Health workers, during their home visits can inquire about the LMP of women and thereby register them as early as possible. The average number of pregnant women that can be expected in a given population can be calculated from the prevailing birth rate of that area e.g. if the birth rate is 25 per thousand population, then 25 women per thousand population are expected to be pregnant at any given point of time.

2. **Regu1ar follow up visits :** Ideally, women must follow up once a month during the first trimester, once a fortnight upto 36 weeks and once a week thereafter. Practically, however, this is not possible for a large proportion of the rural mothers as well as the urban mothers belonging to low socioeconomic groups, who work on a daily wages basis, Studies have shown a direct positive correlation between the number of ANC visits and fetal outcome in terms of birth weight. Under the safe motherhood programme a minimum of three check ups during the pregnancy is recommended.
   a. First check up at 20 weeks or as soon as the pregnancy is known.
   b. Second check up at 32 weeks.
   c. Third check up at 36 weeks.

3. **Anemia Prophylaxis :** All pregnant women should receive 100 tablets of Iron and folic acid during the entire course of pregnancy. Each tablet has 60 milligrams of elemental iron and 500 micrograms of folic acid. The woman should taken one tablet per day as prophylaxis, Those who have anemia should receive two tablets per day for at least three months.

4. **Tetanus Immunization:** The safe motherhood programme aims to eliminate neonatal tetanus. For this, as well as, for the safety of the women, every pregnant women should receive injection T.T. (0.5 cc. IM), the first dose at the first contact and the second dose four weeks later, If the mother has delivered the previous child, less than five years back and had taken two doses of T. T. at that time, then only one dose is given during the present pregnancy.

5. **Safe delivery Practices:** Mothers are encouraged to have institutional deliveries. TBA delivery kits are provided to those mothers who desire to have the delivery at home. The Dais training programme is being conducted in order to train all Dais to adopt safe delivery practices.

   The *Five Clean Practices* are emphasized i.e. clean hands, clean blade, clean cord tie, clean stump and clean delivering surface. Dais delivery kits are equipped with a plastic sheet for conducting the delivery, new blade, sterilized thread, sterilized gauze pieces, soap strips. Delivery kits, packed and pre-sterilized by radiation are available so that the dai does not have to waste time for obtaining the necessary equipment

6. **Strengthening the PHC and referral centres:** The PHCs and referral centres known as FRUs (First Referral Units) are to be strengthened by providing the necessary equipments and instruments to conduct deliveries. FRUs are to be equipped with specialists and instruments for performing LSCS, MTP, Suction and Curettage, vaccum deliveries, blood transfusions etc. Training programme for all levels of staff is to be carried out.

II. STATISTICS

1. Biostatistics

Statistics as taught in PSM is divided into Biostatistics and Vital statistics.

Biostatistics as well as Vital statistics are extremely easy, however due to poor interest shown by the students towards these subjects, they ultimately appear difficult and the students usually mug up the problems for the sake of the examinations. We advise the students to study the following chapter with a little more concentration, since once the problem and the steps to solve them are clearly understood, the student need not mug up problems. Not only this, but once the student understands the application of statistical tests of significance, he will find it useful for future applications for research studies, paper presentations and publications.

Measures of Central Tendency.

If we observe biological values in nature we will notice that, there is usually something called as normal or average value, and all the other observations are clustered closely around this normal or average value.

Example: If you take the systolic BP readings of 100 in 25yr. old individuals, you will notice that most of these are clustered around 110mm of Hg which can be considered as the normal for this age, and few readings will be away from 110mm. There will also be very few readings or an occasional reading that is very much less (e.g. 90mm or 80mm), or very much more (e.g. 150 mm or 180 mm).

These will be considered as extremes, and in medical terminology, as due to pathologic or abnormal states.

This tendency or clustering around the average or normal is called 'Central tendency'.

Central tendency is usually measured by,

i. Arithmetic Mean'or average
ii. Median.
iii. Mode.

Arithmetic Mean

This is a simple average, where you add up all the observations and divide by the lotal number of observations.

e.g: Observations of the Hb or a group of 10 women are

10.2,11.4,8,7.8,8.2,9,10,10.2,10.6,11.

We just add up all these

i.e. 10.2+11.4+8+7.8+8.2+9+10+10.2 +
10.6 + 11 = 96.4.

This is divided by the total number or observations which is 10 in this case

$$\therefore \frac{96.4}{10} = 9.64$$

Hence 9.64 gm of Hb is the Arithmetic mean for this group of observations.

Median

When all the observations are arranged in a ascending or descending order the observation which occupies the central position is the median.

e.g. : Consider the systolic BP of a group of 15 students of the 10th standard of a school.

100mm, 110mm, 106mm, 96mm, 100mm, 90mm, 120mm, 124mm, 110mm, 116mm, 100mm, 106mm, 120mm, 110mm, and 106mm.

First arrange these observations in a descending order.

124 mm
120 mm
120 mm
116 mm
110 mm
110 mm
110 mm
106 mm
106 mm
106 mm
100 mm
100 mm
100 mm
96 mm
90 mm

Now since the total number of observations is odd in number, we notice that the central observation is 106mm of Hg.

Hence the median of this group of observations is,

Median = 106mm Hg.

If the group of observations is even in number e.g. the observations of Hb. of the group of 10 women.

i.e.10.2,11.4,8,7.8,8.2,9,10,10.2,10.6,11,

11.4 We follow the same procedure of arranging
11 the observations in an ascending or descend-
10.6 ing order and then we take the two central ob-
10.2 servations i.e. 10.2 and 10 in this case, add
10.2 them and then divide by 2 (i.e. take the
9 average of the two central observations)
8.2
8
7.8

$$10.2 + 10 = \frac{20.2}{2} = 10.1$$

Hence, the Median = 10.1

For grouped data, where we are given a frequency distribution table we will use the following formula

$$\text{Median} = L + \frac{(n/2 - F)\, e}{f}$$

The central group is called the "median group"

and L = the lower limit of the median group.
n = total No. of observations.
F = total No. of observations upto the median group.
f = total No. of observations in the median group.
e = the class interval of the median group.

e.g.: consider the table for the age wise distribution of 200 cases of TB in a study.

Age	No. of Cases.
0 - 5 yrs.	8
6 - 10 yrs.	15
11 - 15 yrs.	15
16 - 20 yrs.	18
21 - 25 yrs.	12
26 - 30 yrs.	16
31 - 35 yrs.	20
36 - 40 yrs.	24
41 - 45 yrs.	24
46 - 50 yrs.	28
51 - 55 yrs.	20

The median group is (26 - 30yrs.)

L = lower limit of the median group = 26yrs.

n = total no. of observations = 200.

F = total no. of observations upto the median group = 8+15+15+18+12 = 68

f = total no. of observations in the median group = 16.

c = Class interval of the median group = 5 yrs (26-30yrs.) (this class interval includes both 26 and 30yrs. hence it is 5yrs.)

now using the formula.

$$\text{Median} = L + \frac{(n/2 - F)}{f} c$$

$$= 26 + \frac{(200/2 - 68)}{16} 5$$

Hence Median = 36yrs.

Mode

This is the most frequently occurring value in a group of observations.

e.g. : consider the observations of the Hb. of 10 women

8,9,9.6, 10, 10.2, 10,9, 10.2, 10.2, .12.

8
9
9
9.6
10 10
10.2
10.2
10.2
12

First arrange them in an ascending order. You will notice that 10.2 is the most frequently occurring observations.

Hence Mode= 10.2

If we have a group of observations such as,

Hb. 8gm, 9gm, 8.4gm, 9.2gm, 10.4gm, 11gm, 8.6gm, 9.4gm, 10gm, 10.6gm.

we observe that there is no mode or most frequently occurring value. In such cases we can calculate the mode using the formula.

Mode = 3 (Median) - 2 (Mean).

This means that we will have to first calculate both the Mean and the Median and then derive the Mode.

e.g. : Arrange these observations in an ascending order.

8 gm
8.4 gm

$$\text{Median} = \frac{9.2+9.4}{2} = 9.3$$

8.6 gm
9 gm
9.2 gm — Mean = $\frac{94.6}{10}$ = 9.46
9.4 gm
10 gm
10.4 gm — ∴ Mode = (3xMedian)–(2xMean)
10.6 gm — = 3 x 9.3 – 2 x 9.46
11 gm — = 27.9 – 18.92.
∴ Mode = 8.98 gm

In case of frequency distribution tables we can use the formula,

$$\text{Mode} = Lm + \frac{f_1 c}{f_1 + f_2}$$

Where the group which has the maximum number of observations is called the Modal group.

Lm = the lower limit of the Modal group
f_1 = the total number of observations in the Modal group minus the total number of observations in the group prceceding the Modal group.
f_2 = the total no.of observations in the Modal group minus the total no.of observations in the group that follows after the Modal group.
c = The class interval of the Modal group.

e.g .: Let us consider again the table of the age wise distribution of 200 cases of T.B.

Age	No.of cases
0 - 5 yrs.	8
6 - 10 yrs.	15
11 - 15 yrs.	15
16 - 20 yrs.	18
21 - 25 yrs.	12
26 - 30 yrs.	16
31 - 35 yrs.	20
36 - 40 yrs.	24
41 - 45 yrs.	24
46 - 50 yrs.	28
51 - 55 yrs.	20

In this case the gruup which has the maximum observations is (46-50yrs.) i.e. the Modal group.

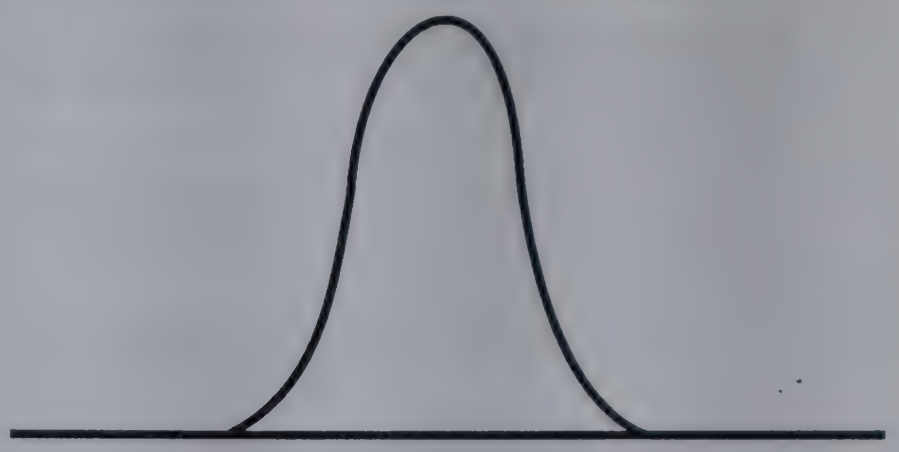
Lm = 46yrs.
f_1 = 28 - 24 = 4
f_2 = 28 - 20 = 8
c = 5 yrs.

Hence Mode = $Lm + \frac{f_1 c}{f_1 + f_2} = 46 + \frac{(4 \times 5)}{4+8} = 46 + 1.66$

Hence Mode = 47.66 yrs. (or 47 yrs. and 8 mths. approx.)

Remember that the Mean, Median and Mode will be the same, only in a standard (normal distribution) curve.

Fig. 1.



: Mean
: Median
: Mode

i.e. if the observations follow the normal or symmetrical distribution.

In case the distribution is asymmetrical then the Mean, Median and Mode will be different

Fig. 2

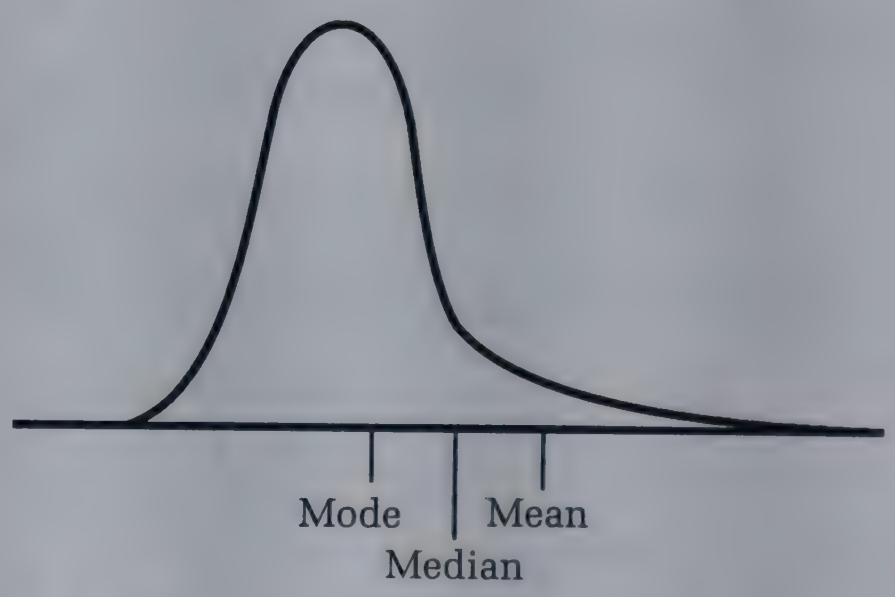
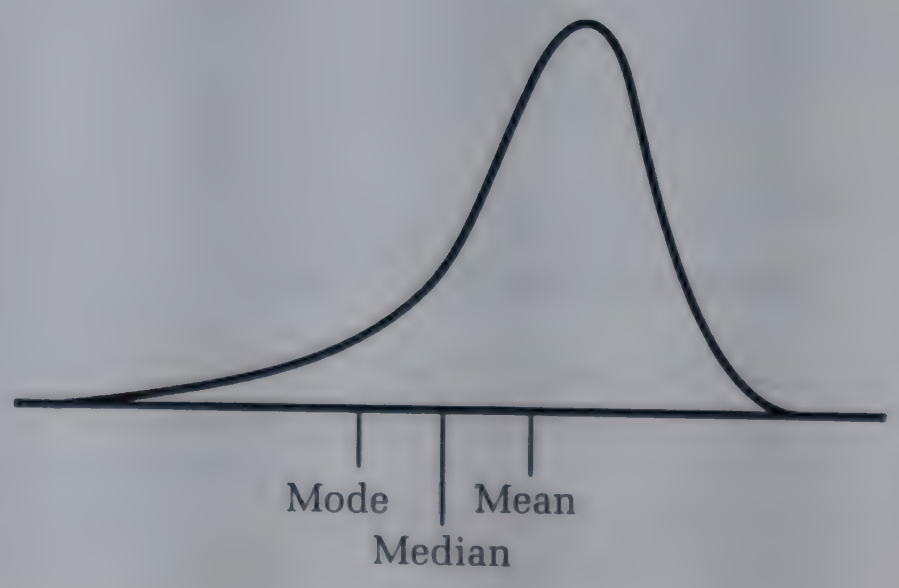


Fig. 3



When and which measure of central tendency is to be used.

The choice of the measure depends on the type of, distribution ie. symmetrical or asymmetrical.

1. In case of symmetrical data, one may use either of the three.
2. a. In case of asymmetrical distribution, the mean is usually unsuitable. When the distribution is more towards the left as in fig. 2 (also known as 'Positive skewing'), then the Mean is more than Median which is in turn more than the Mode.
   b. Where as, when the distribution is more towards the right as in fig.3 (also known as 'negative skewing'), then the Mean is lesser than the Mode. Hence when the distribution is skewed it is preferable to use the Mode or Median.
3. When there are a few observations which are very much away from the normal or centre of the group, then the Mean will get distorted, hence we use the Median in such cases.
   By calculating the Mean and Median or the Mean and Mode. We can say whether the skewing is positive or negative.
   a. If the Median and Mode> Mean it means that the distribution is asymmetrical and has negative skewing e.g. fig.3.
   b. If the Median and Mode < Mean it means that the distribution is asymmetrical and has positive skewing e.g. fig.2.

Measures of Dispersion.

Just as we considered 'Measures of central tendency' i.e. (how close the observations are from the centre i.e. Mean, Median and Mode), similarly we can consider how much each observation is *away* from the centre. This deviation from the centre can be measurcd using 'Measures of deviation or dispersion.

They are,

a. Range.
b. Mean deviation.
c. Standard deviation.

Range.

Range is the interval between the lowest and the highest observation in a group of observations.

e.g. Hb values of 10 women,

8,11.2, 11.4,11.8,6.2,12, 12.8, 10,9.6,10.2.

note that the lowest is 6.2gms.and the highest is 12.8gms.

Hence the range is 6.2gms to 12.8gms.

or 6.6 gms

Mean Deviation

To calculate the Mean deviation, follow these steps:

i. Calculate the arithmetic·Mean ($\bar{x}$)
ii. Find the deviation of each observation from the Mean ($x-\bar{x}$)
iii. Add up all the deviations, *ignoring the Plus/Minus* signs.
iv. Calculate the Mean of these deviations;
i.e. $\frac{\sum (x - \bar{x})}{n}$

e.g. Systolic BP of 10 individuals is

120,140,110,120,130,140,110,120,110,100.

The arithmetic mean is = $\frac{1200}{100} = 120$

x	$\bar{x}$	$(x - \bar{x})$
120	120	0
140	120	+ 20
110	120	- 10
120	120	0
130	120	+ 10
140	120	+ 20
110	120	- 10
120	120	0
110	120	- 10
100	120	- 20
1200		**100**

The total of the deviations = 100

∴ the mean deviation

$= \frac{100}{10}$

M.D. = 10

(ignoring the Plus/Minus signs)

The formula for Mean deviation is

$$M.D = \frac{(x - \bar{x})}{n}$$

x = individual observations X_1, X_2, X3, etc.

$\bar{x}$ = Arithmetic Mean of X_1, X_2, X_3, etc.

n = total number of observations.

The formula for calculating the Mean deviation of frequency distribution table (grouped data) is

$$M.D = \sum f \frac{(x - \bar{x})}{n}$$

f = frequency or the total no. of observations in a group.

n = total no. of observations in all groups

$\bar{x}$ = Arithmetic Mean of the observations

Example for calculating M.D. of grouped data. Consider the age wise distribution 120 TB cases.

Age	no. of cases f	Mid point of each group x	Deviation of the mid point from the mean * $(x - \bar{x})$	$f(x - \bar{x})$
0 - 5 yrs.	8	2.5 yrs	-29	232
6-10	15	7.5	-24	360
11 - 15	15	12.5	-19	285
16 - 20	18	17.5	-14	252
21 - 25	12	22.5	- 9	108
26 - 30	16	27.5	- 4	64
31 - 35	20	32.5	+1	20
36 - 40	24	37.5	+6	144
41 - 45	24	42.5	+11	264
46 - 50	28	47.6	+16	448
51 - 55	20	52.5	+21	420
Total	**200 (n)**			**2597**

(ignoring Plus/Minus signs)

Mean *should first be calculated using the formula

$$\text{Mean} = \frac{\sum fx}{n}$$

Mean = 31.475 (approx. 31.5) in this case.

$$\text{M.D} = \frac{\sum f(x - \bar{x})}{n} = \frac{2957}{200} = 12.985$$

M.D. = 12.985 (approx. 13 yrs)

Standard Deviation

To calculate the S.D follow these steps:

i. Calculate the arithmetic Mean $(\bar{x})$

ii. Find the deviation of each observation from the Mean i.e. $(x - \bar{x})$

iii. Calculate the square of these deviations; i.e. $(.x - \bar{x})^2$

iv. Add up all these squares $\sum (x - \bar{x})^2$

v. Divide by (n - 1)
(n) total no.of observations ;

$$\frac{\sum (x - \bar{x})^2}{(n - 1)}$$

vi. S.D = square root of this.

i.e. $\sqrt{\frac{\sum (x - \bar{x})^2}{(n - 1)}}$

$$\therefore \text{S.D} = \sqrt{\frac{\sum (x - \bar{x})^2}{(n - 1)}}$$

Take the same example of the systolic BP of 10 individuals,

120, 140, 110, 120, 130, 140, 110, 120,110, 100mm Hg.

The Arithmetic Mean = 120mm Hg.

Arrange the observations in a table form as follows.

	x	$x - \bar{x}$	$(x - \bar{x})^2$
	120	0	0
	140	+ 20	400
	110	– 10	100
	120	0	0
	130	+ 10	100
	140	+ 20	400
	110	– 10	100
	120	0	0
	110	-10	100
	100	- 20	400
Total	**1200**		**1600**

$$[\sum (x - \bar{x})^2] = 1600$$

n = total no. of observations = 10

$$\therefore \text{S.D} = \sqrt{\frac{(x - \bar{x})^2}{(n - 1)}} = \sqrt{\frac{1600}{9}} = \sqrt{177.77}$$

$$\therefore \text{S.D} = 13.33$$

*** Remember that when the total no. of observations (n) is very large then we may use the formula.**

S.D $= \sqrt{\frac{\sum f\,(x-\bar{x})^2}{n}}$ instead of S.D. $= \sqrt{\frac{\sum f\,(x-\bar{x})^2}{n-1}}$

depending upon the size of the total no.of observations (the sample size). or (n).

To calculate the S.D in case of a frequency distribution table (grouped data) we may use the formula

$$S.D = \sqrt{\frac{\sum f\,(x-\bar{x})^2}{n}} \quad \text{or S.D.} = \sqrt{\frac{\sum f\,(x-\bar{x})^2}{n-1}}$$

f = frequency or the total no. of observations in a group

n = total no. of observations

x = mid point of each group

$\bar{x}$ = arithmetic mean of the observations

Example: Age wise distribution of 200 cases of T.B.

Age	no. of cases f	Mid pt. of each group x	Deviation of the mid pt. from the mean* $(x-\bar{x})$	$(x-\bar{x})^2$	$f(x-\bar{x})^2$
0 - 5 yrs.	8 persons	2.5 yrs.	– 29	841	6728
6 - 10	15	7.5	– 24	576	8640
11 - 15	15	12.5	– 19	361	5415
16 - 20	18	17.5	– 14	196	3528
21 - 25	12	22.5	– 9	81	772
26 - 30	16	27.5	– 4	16	256
31 - 35	20	32.5	+ 1	1	20
36 - 40	24	37.5	+ 6	36	2064
41 - 45	24	42.5	+ 11	121	2904
46 - 50	28	47.5	+16	256	7168
51 - 55	20	52.5	+ 21	441	8820
Total	**200 (n)**				**46315**

$\sum [f(x-\bar{x})^2] = 46315$

Mean* should first be calculated using the forrnula

$$\text{Mean} = \frac{fx}{n}$$

Mean = 31.475 (approx. 31.5) in this case.

$$\text{S. D.} = \sqrt{\frac{\sum f\,(x-\bar{x})^2}{n-1}} = \sqrt{\frac{46315}{(200-1)}} = \sqrt{\frac{46315}{199}} = \sqrt{232.74} = 15.256$$

$\therefore$ S.D = 15.256

Please note that S.D is denoted as just (S) or (O) is some books.

STATISTICAL TESTS OF SIGNIFICANCE

Students are expected to demonstrate the use of,

1. Standard error of Mean.
2. Standard error of Proportion.
3. Standard error of difference of Means.
4. Standard error of difference of Proportions.
5. Chi square test.
6. t test

Let us see when and how each of these tests are to be used.

In solving problems involving tests of significance we follow these steps.

1. Note down the given data.
2. Formulate in clear words the question to be answered.
3. Decide on the basis of the first two steps as to which test is to be used.
   a. If the sample Mean has to be compared with the Mean of the population or universe (ie. the larger group from which the sample is drawn), we will, apply the test,'Standard error of Mean'.
   b. If two samples are to be compared and their Means are given, then we will apply the test 'Standard error of difference of Means'.
   c. If the sample is to be compared with the group from which the sample is drawn (i.e. population or universe) and the proportion (percentages) are given instead of Means, then we will apply the test 'Standard error of Proportion.'
   d. If two samples are to be compared and instead of their Means their proportions (percentages) are given, then we will apply the test 'Standard error of difference of Proportions'
   e. If the Means or proportions of two samples are to be compared in case of small samples, then we can use 't' test (also known as student's 't' or Gosset's 't' test). (Remember that if 't' test is to be applied the student is either supplied with a 't' table or given the relevant values from a 't' table, along with the other given data).
   f. To compare two samples whose proportions or actual no. of cases are given we can also employ a chi square (x^2) test (Remember again that in case a x^2 test is required to be applied, the student is either supplied with a x^2 table or given the relevant extracts from the x^2 table along with other given data). x^2 test is usually used in case control and cohort studies to compare the effect/influence of the causative factor on the cases (or cohorts) and the control group. e.g. influence of smoking on lung cancer.
4. Once the test to be used is decided then write down its formula.
5. Now look for all the values required in the " formula, from the data you have noted down. Put the required values in the right parts of the formula and proceed to solve the problem.

Remember:

   a. All steps should be properly noted down.
   b. Do not forget to write the full answer, don't just write in numericals.
   c. If by chance the values required by the formula are not available in the given data, your first guess should be that you have selected the wrong test for application.

In this case you have to first reconsider the test to be applied. If you still think that it is appropriate, then, look again at the given data and find out whether the missing value required for the formula can be derived from parts of the given data.

Let us now proceed to illustrate these steps with the help of examples.

Standard Error of Mean.

Example 1.

The Hb. of 100 women was studied. It showed a Mean of 10gm%with a S.D of 1.5gm%. Can you say whether these women were drawn from a rural area if the Mean Hb for rural Indian women is 11gm%.

Answer

Let us proceed as per the steps outlined earlier.

Step: 1.

Given data :

n (total no. of women in the sample) = 100

(Ms) sample Mean = 10gm%

(S.Ds) sample S.D = 1.5gm%

(Mu) Mean of the Universe = 11gm%

(in this case the rural Indian women, from which the sample is supposed to have been drawn, is the Universe)

Step : 2.

Question to be answered:

Whether the given sample of 100 women is drawn from the Universe, (Rural Indian women).

Step: 3.

Which test is to be applied.

This problem involves the comparison of the sample Mean with the Mean of the population/ universe hence we have to apply the test 'Standard error of Mean' .

Step: 4.

Formula for the test to be applied.

$$\text{Std. error of Mean (S.E.M)} = \frac{\text{S.D}}{\sqrt{n}}$$

Step: 5.

Fill the required values into the formula, from the given data

$$\text{S.E.M} = \frac{1.5}{\sqrt{100}} = \frac{1.5}{10} = 0.15$$

Now it is known that, if the sample is presumed to be drawn from the said universe, then the Mean of the sample should lie within a range of +2 S.E.M &-2 S.E.M. from the Mean of the universe.

(At 95% confidence limits)
or 5% error level

Hence.

The Universe Mean (Mu) ± 2 S.E.M = 11 ± 2 x 0.15
= 1 ± 0.30
= 10.7 to 11.3 gm%

The sample Mean (Ms) = 10gm% lies outside, this range i.e. outside Mu ± 2 S.E.M i.e. 10.7 to 11.3gm%

Hence the conclusion is that the sample is not drawn from the universe i.e. rural Indian women.

Please note that if the S.D of the universe is given, instead of the S.D. of the sample, as given in this example, we will still follow the same procedure.

Example: 2. Standard error of Difference of Means.

The Mean height of 100, 18year old boys was 160cms, With a standard deviation of 7 cms. Another group. of 200, 18 year old boys was 168 cms, with a standard' deviation of 8 cms. Do you think that both these samples were drawn from the same universe.

Answer

Step: 1.

Given data:

Mean ht. of the 1st sample, (M_1) = 160 cms.
S.D. of the 1st sample, $(S.D_1)$ = 7 cms.
total size of 1st sample, (n_1) = 100
Mean ht.of 2nd sample (M_1) = 168 cms.
S.D of 2nd sample $(S.D_2)$ = 8 cms.
total size of 2nd sample (n_2) = 200

Step: 2.

Question to be answered :

Whether both samples are drawn from the same universe.

Step: 3.

Which test is to be applied: .

This problem involves the comparison or the means of two different samples. Hence the test to be applied is 'Standard error of difference of Means.'

Step: 4.

The formula of the test.

$$\text{S.E. (Difference of Means)} = \sqrt{\frac{(S.D_1)^2}{n_1} + \frac{(S.D_2)^2}{n_2}}$$

Step: 5.

Fill the required values in the formula,

$$\therefore \text{S.E. (Diff of means)} = \sqrt{\frac{(7)^2}{100} + \frac{(8)^2}{20}} = 0.9$$

$\therefore$ 2 S.E = 2 x 0.9 = 1.8 cms

M_1 = 160 cms. M1 ± 2 S.E (Difference of Means)
= 160 ± 1.8
= 158.2 to 161.8

M_2 = 168 cms, does not fall within the range M± 2 S.E
i.e. within 158.2 cms. to 161.8 cms.

Hence the conclusion is that both these samples are not drawn from the same universe, if the confidence limits are set at 95% level.

Example: 3. Standard Error of Proportion

As per available statistics, it is known that 80% of pregnant women in India are anemic. A study of 200 pregnant women showed that 75% were anemic. Is it possible that these pregnant women were Indian.

Answer

Step: 1.

Given data:

Size of sample (n) = 200

Proportion (%) of anemic women (p) in the sample. = 75%

Proportion of anemic women among pregnant Indian women (P) = 80%

Step: 2.

Question to be answered.

Whether the sample is drawn from the universe, pregnant Indian women.

Step: 3.

Which test to be applied:

This problem involves the comparison of the proportion of anemics from the sample with the proportion of anemics from the universe i.e. pregnant Indian women. Hence the test to be applied is Standard Error of Proportion.

Step: 4.

Formula:

$$\text{S.E of proportion (S.E.P)} = \sqrt{\frac{pq}{n}}$$

Where p = Proportion of diseased (anemic in this case) and

q = Proportion of not diseased (i.e.100 - P)

n = total no. persons in the sample (sample size)

Step: 5.

Fill the required values in the formula.

$$\therefore \text{S.E.P.} = \sqrt{\frac{pq}{n}} = \sqrt{\frac{80 \times 20}{200}} = 2.8$$

$\therefore$ S.E.P = 2.8

$\therefore$ 2 S.E.P = 5.6

$\therefore$ P ± 2 S.E. = 80 ± 5.6

= 74.4 to 86.6

The proportion of anemics in the sample = 75% falls within this range ie. within 74.4 to 86.6

Hence the conclusion is that the sample of pregnant women studied were drawn from the universe Indian women. (At 95% confidence limits)

Please note that : Sometimes the actual number of cases are given, in such problems we can calculate the (%) proportion first and then proceed to solve the problem.

Example: 4. Standard Error of Difference of Proportion.

Anthropometric studies of nutritional status of 200 children (below 6yrs. of age) were carricd out and results showed that nearly 40% of the children were suffering from various grades of Malnutrition. Similar studies of another group of 80 children showed that only 16 children were malnourished. Can we say that both these samples were drawn from the same socio-economic group.

Step: 1.

Given data:

Sample size of group 1, (n_1) = 200

proportion of malnourished Children in group 1, (P_1) = 40%

Sample size of group2 (n_2) = 80

no.of malnourished children in group 2, = 16

$\therefore$ the proportion of Malnourished Children in group2 (P_2) = 20%

since P_1 and P_2 are given we can derive (q_1)

(q_1) (proportion of normal children in group 1) = (100 - P_2) = (100 – 40) = 60%

(q_1) (proportion of normal children in group 2) = (100 – P_2) = (100-20) = 80%

Step: 2.

Question to be answered

Whether sample one is comparable.: with sample two as regards, the proportion of malnourished children ill in these groups.

Step: 3.

Test to be applied.

In this case the proportion of malnourished children in group 1 are to be compared with the proportion of malnourished children in group 2, hence the test to be applied is, 'Standard error of difference of proportion'.

Step: 4.

Formula of the test.

$$\text{S.E. (Difference of proportion)} = \sqrt{\frac{p_1q_1}{n_1} + \frac{p_2q_2}{n_2}}$$

Step: 5.

Fill the required values in the formula.

$\therefore$ 2 S.E. (Diff. of proportion)

$$= \sqrt{\frac{40 \times 60}{200} + \frac{20 \times 80}{80}} = 5.65$$

$\therefore$ 2 S.E = 2 x 5.65 = 11.3

$\therefore$ P_1 (±) 2 S.E = 40 (±) 11.3 = 28.7% to 51.3%

P_2 = 20% does not lie within this range
ie. within 28.7% to 51.3%

Hence the conclusion is that these samples are not drawn from the same universe (socioecomomic group in this case) at 95% confidence limits.

Example: S. Chi Square. test

After the introduction of a new chemical process in a fertilizer industry it was noticed that some workers who were handling the concerned chemical were complaining of dermatitis of hands. On the demand, for compensation by the workers union the management carried out a study.

The results showed that out of 200 workers employed in the concerned unit, 8 were suffering from dermatitis, and out of 120 workers working in another unit which was not handing the incriminated chemical, 2 workers were suffering from similar dermatitis. What is your conclusion as regards the relationship between the chemical and dermatitis.

Student is provided with a x^2 table.

Answer:

Step: 1.

Given data:

Total no.of workers in the chemical unit = 200

no.of workers from this unit suffering from dermatitis = 8

∴ no. of workers from this unit, not suffering = 192

total no.of workers in'the other unit = 120

no.of workers in the other unit suffering form, dermatitis = 2

∴ no. of workers not suffering in this unit = 118

Step: 2.

Question to be answered

Is the chemical responsible for the dermatitis or not.

Step: 3.

The test to be applied.

The relationship between the cause and the effect is to be tested from among cases and controls. Hence chi square test is to be applied.

Step: 4.

Formula

$$X^2 = \frac{(O - E)^2}{E}$$

Where O = Observed value.
and E = Expected value.

Observed values are given, Expected values can be calculated using the formula

$$E = \frac{\text{Horizontal total x vertical total}}{\text{grand total.}}$$

Step: 5.

Fill the required values in the formula:

In case of x^2 test we follow this procedure

(a). first prepare a table like this.

unit.	dermatitis cases	non-cases	Total
Chemical using unit	8 (6.25)	192 (193.75)	200 (200)
other unit	2 (3.75)	118 (116.25)	120 (120)
Total	**10** (10)	**310** (310)	**320** (320)

This is called a classical 2 x 2 table. The observed values are given in the table

(b) Now we have to calculate the expected values for each cubicle.

$$E = \frac{\text{Horizontal total x Vertical total}}{\text{grand total.}}$$

i) ∴ E-value for the cubicle where the observed value is 8 will be

$$E = \frac{200 \times 10}{320} = 6.25$$

ii) ∴ E-Value for the cubicle where the O value is 2 is $E = \frac{120 \times 10}{320} = 3.75$

iii) ∴ E-value for the cubicle where the O value is 192 is $E = \frac{200 \times 310}{320} = 193.75$

iv) ∴ E-value for the cubicle where the O value is 118 is $E = \frac{120 \times 310}{320} = 116.25$

The expected values are shown in brackets in the table.

Please note that, in the table, the totals of the expected values (shown in brackets) should be the same as that of the observed values. This is a useful counter check to see that your calculations of the E values are correct.

c) We have to now calculate the x2 values for each cubicle. $x^2 = \frac{(O - E)^2}{E}$

i) ∴ x^2 for the cubicle with O value 8 and E-value 6.25. $x^2 - = \frac{(8 - 6.25)^2}{6.25} = 0.49$

ii) ∴ x^2 for the cubicle with O value 2 and E-value 3.75. $x^2 = \frac{(2 - 3.75)^2}{3.75} = 0.82$

iii) ∴ x^2 for the cubicle with O value 192 and E value 193.75. $x^2 = \frac{(192 - 103.95)^2}{193.75} = 0.02$

iv) ∴x^2 for the cubicle with O value 118 and E value 116.25. $x^2 = \frac{(118 - 116.25)^2}{116.25} = 0.03$

d) Now add up the x^2 values for all the four cubicles, which will give the x^2, value for the whole table.

∴ $x^2 = 0.49 + 0.82 + 0.02 + 0.03$
$x^2 = 1.36.$

e) Calculate the degree of freedom (Df). This is given by the formula.

$(H - 1) \times (V - 1)$

Where H = total no. of Horizontal columns.
V = total no. of Vertical columns.
In this problem H = 2
and V = 2
Hence Df = $(2 - 1) \times (2 - 1) = 1 \times 1 = 1$

Now refer to the given x^2 table and place the calculated x^2 value of the problem (ie.1.36) in the x^2 table against the calculated degree of freedom (ie. 1 in this case).

You will notice that the x^2 value in this case lies towards the left of the P value .05. The statistical interpretation in this case is *not significant.* Which in terms of the conclusion for this problem means that the occurrence of dermatitis in the two groups is *not significantly different.* Hence the concerned chemical cannot be held responsible for the occurrence of the dermatitis.

Please note that in a x^2 table

i) The F column denotes the degree of freedom.
ii) The probability of occurrence is given by the columns below the values.
.50, .10, .05, .02, .01, .001 etc.
iii) x^2 values lying towards the left of the .05 column should be considered as not significant.
iv) x^2 values lying towards the right of the .05 column should be considered as significant (those lying further away towards the right e.g. .01 or .001 columns should be considered as highly significant).
v) Where the interpretation is significant the conclusion is that there is a relationship between the cause and the effect.
vi) Where the interpretation is *not significant* the conclusion is that *there is no relationship between the cause and the effect.* (In the problem just showed the cause was the chemical and the effect was the dermatitis. On studying the cases and controls (non-cases) with and without exposure to the chemical, we found that the x^2 value is not significant and hence we came to the conclusion that there is no relationship between the cause (the chemical) and the effect (the dermatitis)

Table of values of X^2 corresponding to different levels of significance

d.f.	P					
	0.50	.10	.05	.02	.01	.001
			Range of significance			
1	0.46	2.71	3.84	5.41	6.63	10. 83
2	1.39	4.61	5.99	7.82	9.21	13.83
3	2.37	6.25	7.82	9.84	11.34	16.27
4	3.36	7.78	9.49	11.67	13.28	18.47
5	4.35	9.24	11.07	13.39	15.09	20.52
6	5.35	10.65	12.59	15.03	16.81	22.46
7	6.35	12.02	14.07	16.62	18.48	24.32
8	7.34	13.36	15.51	18.17	20.09	26.13
9	8.34	14.68	16.92	19.68	21.67	27.88
10	9.34	15.99	18.31	21.16	13.21	29.56
11	10.34	17.28	19.68	22.62	24.72	31.26
12	11.34	18.55	21.03	24.05	26.22	32.91

P = Probability of getting a larger value of XZ by chance alone

At this stage it would be useful for the student to understand that, 95% confidence limits or 5% error level is, by standard convention, used for determining the level of significance in all tests of significance and is approximately equal to 2 x S. E. Hence ± 2 S.E has been used in previous examples. And hence in chisquare and 't' tables values towards the left of the .05 (5%) cloumn are considered as not significant, while values from .05 towards the right are considered as significant. The investigator/experimenter is free to set the error level at 10%, 5% or 1 % or whatever level he thinks fit as per the study under consideration. Students may however use 5% error level for their calculations routinely.

Example: 6. 't' test

On routine school health check up, the height of 25 boys of the 4th standard showed a mean height of 39 inches with a standard deviation of 4 inches. Can you say that these boys were from a rural primary school if it is known that the mean height of 4th standard boys from rural India is 32 inches. 't' table is given.

Answer :

Step: 1.

Given data.

Sample size (n)	=	25
Sample mean (x)	=	39 inches
Sample S.D (S.D)	=	4 inches
(Universe) population mean (μ) of rural 4th standard boys.	=	32 inches

Step: 2.

Question to be answered.

Whether these boys belong to a rural school.

Step: 3.

Test to be applied

The problem involves the comparison of the sample mean with the universe / population mean. Hence we can apply the test Standard Error of Mean. However since the sample size is below 30 in this case, we apply the 't' test.

Step: 4.

The formula of the 't' test

$$\text{'t'} = \frac{(x - \mu)}{S.D} \times \sqrt{n}$$

Step: 5.

Substituting the given data into the formula.

$$\therefore\ t = \frac{39 - 32}{4} \times \sqrt{25} = 1.75 \times 5 = 8.75$$

$$\therefore\ t = 8.75$$

Since there is only one sample the degrees of freedom in this case is given by

$$d.f = (n - 1) = (25 - 1) = 24.$$

Check the P value in the given 't' table against the d.f 24 you will notice that 't' = 8.5 at d.f 24 lies towards the right of the 0.05 column.

$\therefore$ the conclusion is that there is a significant difference between the two means.

$\therefore$ The sample studied is not drawn from Indian rural school boys of the 4th standard.

Example :7. Unpaired 't'

During an anthropometric survey of nursery class children in Bombay, it was noticed that one group of 25 children from school A, showed a mean weight of 12kg, with a S.D of 1.5kg, while another group of 16 children from school B, showed a mean weight of 10kg, with a S.D of 1.8kg. Is it possible that both these groups were drawn from the same socio-economic class.

Answer :

Step: 1.

Given data:

Sample size of gr. A (n_1)	=	25
Mean wt. of sample A ($\bar{x}_1$)	=	12 kg.
S.D of sample A ($S.D_1$)	=	1.5 kg.
Sample size of gr. B (n_2)	=	16
Mean wt. of sample B ($\bar{x}_2$)	=	10 kg.
S.D of sample B ($S.D_2$)	=	1.8 kg.

Step: 2.

Question to be answered:

Whether sample A and sample B belong to same socioeconomic class.

Step: 3.

Test to be applied:

Since this problem involves the comparison of the means of two independent samples, we can apply the test Standard Error of difference of Means. However, since the sample sizes are less than 30, we will apply the 't' test. And the two sets of observations are not paired observations, hence we will apply 'unpaired t' test.

Step: 4.

Formula of the test.

$$\text{(unpaired) } t = \frac{x_1 - x_2}{S.D} \times \sqrt{\frac{n_1 n_2}{n_1 n_2}}$$

SO = combincd standard deviation of both the samples.

In this case thc combined S.D required in the formula for 't' test is given by the formula.

$$S.D = \sqrt{\frac{(n_1 - 1)\, S.D_1^2 + (n_2 - 1)\, S.D_2^2}{n_1 + n_2 - 2}}$$

Degrees of freedom (Of) = $(n_1 + n_2 - 2)$

Step: 5.

Substituting the required valucs in the formula we get,

$$\therefore \text{ S.D } \sqrt{\frac{(25-1)\,(1.5)^2 + (16-1)\,(1.8)^2}{25 + 16 - 2}} = 1.62$$

$$\therefore \quad t = \frac{12 - 10}{1.62} \times \sqrt{\frac{25 \times 16}{25+16}} = 3.84$$

$$\therefore \quad t = 3.84$$

degrees of freedom = (25 + 16 – 2) = 39

Check the P value in the 't' table against the degrees of freedom 39.

You will notice that the 't' value ie. 3.84 lies towards the right of 0.05 column, i.e. the difference is significant, i.e. both samples are not drawn from the same universe.

∴ Both samples do not belong to the same socioeconomic class.

Paired 't'

't' test for the difference between the Means of two sets of (dependent or paired)observations.

$$t = \frac{\bar{d}\sqrt{n}}{S.D}$$

d = Mean Increase

$$S.D = \sqrt{\frac{\Sigma\,(d - \bar{d})^2}{n-1}}$$

Example : 8

11, school boys included in a 'Health Education Program' were given a pretest and a post-test. Their marks at these tests were as follows.

Boy no.	1	2	3	4	5	6	7	8	9	10	11
Pretest marks	23	20	19	21	18	20	18	17	23	16	19
Post-test marks	24	19	22	18	20	22	20	20	23	20	17

Is there any significant change in the knowledge of these boys after the 'Health Education Programme' ?

Answer :

Step: 1.

Given data:

Two sets of marks of 11 boys as mentioned above.

Step: 2.

Question to be answered:

Are the marks of the pre and post-test significantly different?

Step: 3.

Test to be applied:

't' test for finding out the difference in the Means of two paired samples.

Step: 4.

Formula:

$$t = \frac{\bar{d}\sqrt{n}}{S.D} \quad \& \text{ S. D. } = \sqrt{\frac{\Sigma\,(d - d)^2}{n - 1}}$$

degree of freedom. df = (n – 1)

Step: v. — Substituting the values in the Cannula:
First make a table.

Boy no.	Pretest	Post-test	difference (d)	$d-\bar{d}$	$(d-\bar{d})^2$
1	23	24	1	0	0
2	20	19	-1	- 2	4
3	19	22	3	+2	4
4	21	18	-3	- 4	16
5	18	20	2	+1	1
6	20	22	2	+1	1
7	18	20	2	+1	1
8	17	20	3	+2	4
9	23	23	0	- 1	1
10	16	20	4	+ 3	9
11	19	17	-2	-3	9
TOTAL			**11**		**50**

$\Sigma d = 11.$

$\therefore \bar{d} = \frac{11}{11} = 1$

$\Sigma (d-\bar{d})^2 = 50$

$n = 11$

$$S.D = \sqrt{\frac{\Sigma (d-\bar{d})^2}{(n-1)}} = \sqrt{\frac{50}{11-1}} = \sqrt{\frac{50}{10}} = 2.236$$

$$t = \frac{\bar{d}\sqrt{n}}{S.D.} = \frac{1\sqrt{11}}{2.236} = \frac{1x3.32}{2.236} = 1.48$$

d. f $= (n-1) = (11-1) =$ 10

Looking at the 't' table at degree of freedom 10 you will notice that the value of t = 1.48 lies to the left of the 0.05 column, which means that the difference is not significant.

$\therefore$ the boys have not benefited significantly from the health education program.

Table of t - Distribution

Degrees of Freedom				Significant Range			
(df)	.3	.2	.1	.05	.02	.01	.001
1	1.963	3.078	6.314	12.706	31.821	63.657	636.619
2	1.386	1.886	2.920	4.303	6.965	9.925	31.598
3	1.250	1.638	2.353	3.182	4.541	5.841	12.941
4	1.190	1.533	2.132	2.776	3.747	4.604	8.610
5	1.156	1.476	2.015	2.571	3.365	4.032	6.859
6	1.134	1.440	1.943	2.447	3.143	3.707	5.959
7	1.119	1.415	1.895	2.365	2.998	3.499	5.405
8	1.108	1.397	1.860	2.306	2.896	3.355	5.041
9	1.100	1.383	1.833	2.262	2.821	3.250	4.781
10	1.093	1.732	1.812	2.228	2.764	3.169	4.587
11	1.088	1.363	1.769	2.201	2.718	3.106	4.437
12	1.083	1.356	1.782	2.179	2.681	3.055	4.318
13	1.079	1.350	1.771	2.160	2.650	3.012	4.221
14	1.076	1.345	1.761	2.145	2.624	2.977	4.140
15	1.074	1.341	1.753	2.131	2.602	2.947	4.073
16	1.071	1.337	1.746	2.120	2.583	2.921	4.015
17	1.096	1.333	1-.740	2.110	2.567	2.898	3.965
18	1.067	1.330	1.734	2.101	2.552	2.878	3.922
19	1.066	1.328	1.729	2.093	2.539	2.861	3.883
20	1.064	1.325	1.725	2.086	2.528	2.845	3.850
21	1.063	1.323	1.721	2.080	2.518	2.831	3.819
22	1.061	1.321	1.717	2.074	2.508	2.819	3.792
23	1.060	1.319	1.714	2.069	2.500	2.807	3.767
24	1.059	1.318	1.711	2.064	2.492	2.797	3.745
25	1.058	1.316	1.708	2.060	2.485	2.785	3.725
30	1.055	1.310	1.697	2.042	2.457	2.750	3.646
40	1.050	1.303	1.684	2.021	2.423	2.704	3.551
60	1.046	1.296	1.671	2.000	2.390	2.660	3.460
100	1.043	1.293	1.660	1.984	2.364	2.641	3.390
120	1.041	1.289	1.658	1.980	2.358	2.617	3.373
α	1.306	1.282	1.645	1.960	2.326	2.575	3.290

2. Vital statistics

These are numerical data regarding vital events in the community (morbidity, mortaliy and fertility) and methods of drawing inference from these data. Students of PSM are expected to know certain definitions and be able to ealculate certain fertility (demographic), morbidity and mortality rates.

These are discussed here along with appropriate examples wherever required.

Fertility statistics.

Crude Birth Rate (CBR) is defined as

$$\text{CBR} = \frac{\text{total no.of live births in a year in a defined geographic area}}{\text{Mid year population of that geographic area}} \times 1000$$

and is written as eg.

CBR = 25 per 1000 population.

General Fertility Rate (GFR)

$$\text{GFR} = \frac{\text{Total no.of live births in a given year in a delined geographic area}}{\text{Mid year female population in the reproductive age group (15-45yrs.) from the same area and during the same year.}} \times 1000$$

Illustration 1 : In a small town with mid yr. population 300,000 the live births in the year 1991 were 7500. The no.of females in the reproductive age group (ie. 15-45years) in this town were 100,000 in the year 1991 Calculate the Crude birth rate and the General Fertility Rate.

$$\text{CBR} = \frac{\text{total live births}}{\text{Mid yr. population}} \times 1000$$

$$= \frac{\cancel{7500}^{25}}{\cancel{300,000}} \times \cancel{1000} = 25 \text{ per } 1000 \text{ population}$$

$$\text{GFR} = \frac{\text{total live births}}{\text{Mid yr. 15-45yr. female popu.}} \times 1000$$

$$\frac{\cancel{7500}^{75}}{100,000} \times \cancel{1000} = 75 \text{ per } 1000 \text{ (15-45) female population}$$

Note:

i. When the Mid year population is not elearly stated as such, consider the given population as mid year population for the purpose of calculation.

ii. Always remeber to write your answer in. full.
eg. in the above illustration.

CBR = 25 would be only part of the answer

You should write it fully as

CBR = 25 per 1000 population.

iii. You must have also realised that following the same steps as we did in Biostats would help in making the problem simple and clear.

Recapitulating .the steps.

Step: 1. Write down the given data.
Slep: 2. State the Question to be answered.
Step: 3. (Since there is no test to be applied), we can directly write the formula.
Step: 4. Write down the given values in the formula and proceed to answer the problem.

Measurement of Mortality

1. Crude death rate is defined as :

$$\text{CDR} = \frac{\text{Total no.of deaths in a year in a defined geographic area}}{\text{The Mid yr population in the same area}} \times 1000$$

2. Infant mortality rate is defined as:

$$\text{IMR} = \frac{\text{No.of deaths of children below 1 yr. age during a given year, in a defined geographic area}}{\text{No of live births, in the same year, in the same area.}} \times 1000$$

3. Neonatal mortality rate is defined as :

$$NMR = \frac{\text{No of deaths of children below 1 month of age, in a given year, in a defined geographic area}}{\text{No of live births, in the same year, in the same area.}} \times 1000$$

4. Perinatal Mortality rate is defined by the formula.

$$PMR = \frac{\text{No of late fetal deaths (after 28 weeks of gestation) + death of children below 1 wk. of age.}}{\text{Total no.of births (still births + live births)}} \times 1000$$

5. Maternal mortality rate is defined by the formula.

$$MMR = \frac{\text{No.of deaths from causes directly related to Pregnancy and puerperium}}{\text{No. of live births in the same year.}} \times 1000$$

Illustration 2 .

In a town with population 800,000, some of the vital statistics data available are as under. For the year 1991, total no.of deaths are 8000, total live births 20,000, total still births 400, total no.of deaths below 1 wk. is 200, total no.of deaths below 1 month is 800 , total no.of deaths below 1yr. of age is 2000, no.of deaths of mothers due to pregnancy and puerperal causes is 80. Calculate, the IMR, NMR, PMR, COR, MMR, for the year 1991 for the town.

Step 1:

Given data is:

Mid year population	=	800,000.
Total no. of deaths	=	8,000.
Total live births	=	20,000.
Total still births	=	400.
Total deaths < 1 week	=	200.
Total deaths < 1 month	=	800.
Total deaths < 1 year	=	2000.
Total deaths due to Pregnancy and puerperium	=	80.

Step 2:

Question to be answered is, Calculate the I MR, NMR, PMR, COR and MMR

Step 3 and 4 :

Formula and solution:

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1) $$IMR = \frac{\text{deaths} < 1\text{ yr}}{\text{live births}} \times 1000 = \frac{100}{\cancel{20000}} \times \cancel{1000} = 100$$

$\therefore$ IMR = 100 per 1000 live births

2) $$NMR = \frac{\text{death} < 1\text{mth}}{\text{live births}} \times 1000 = \frac{40}{\cancel{20000}} \times \cancel{1000} = 40$$

$\therefore$ NMR = 40 per 1000 live births

---

3) $$PMR = \frac{\text{total still births + deaths below 1 wk}}{\text{total live births + still births}} \times 100 = \frac{400 + 200}{20{,}000 + 400} \times 1000$$

$$= \frac{600}{20400} \times 1000 = 29.4$$

$\therefore$ PMR = 29.4 per 1000 births (live + still)

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4) $$CDR = \frac{\text{Total deaths}}{\text{population}} \times 1000 = \frac{\cancel{8000}}{\cancel{800{,}000}} \times \cancel{1000} = 10$$

CDR = 10 per 1000 population

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5) $$MMR = \frac{\text{deaths due to pregnancy and peurperal causes}}{\text{live births}} \times 100 = \frac{80}{\cancel{20000}} \times \cancel{1000} = 4$$

$\therefore$ MMR = 4 per 1000 live births

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3. Assessment of Nutritional Status - By Anthropometry

DEFINITION:

Nutritional anthropometry is the measurement of human body at various ages and levels of nutritional status.

MEASUREMENTS:

The standard measurement taken during routine surveys are:

(1) Body Weight
(2) Crown - Heel Length or Standing Height
(3) Mid-Upper Arm Circumference and
(4) Fat Fold at Triceps.

Circumference of Head and Chest are also included in surveys of children under five years of age.

BODY WEIGHT:

It is very sensitive to even minor changes in nutritional status & in diseases like measles and diarrhoea.

Technique:

A beam or lever actuated scales, with an accuracy of 50-100 gms. should be preferred for weighing. The commonly used bathroom weighing scales are not recommended as the measurements are not accurate because the spring can loose its tension due to regular use and changes in weather conditions. Studies by the NIN at Hyderabad indicate that errors upto 1.5 kg in weights of individuals can occur with bathroom scales as compared to the weights of the same individuals taken with lever actuated balances. These errors can drastically change the diagnosis in young children. Beam balances, have been found to be reliable and are extensively used in ICDS projects.

The following precautions should be taken while measuring body weight:

1. The zero error of the weighting scale should be checked before taking the weight and corrected if required.
2. The individual should wear minimum clothing, and be without shoes or chappals.
3. The individual should not lean against or hold anything, while the weight is being recorded.
4. The measurements, should preferably be taken under basal conditions in early mornings.

HEIGHT :

The height of an individual is influenced by hereditary as well as environmental factors. The maximum growth potential of an individual is decided by hereditary factors, while the environmental factors like nutritional status and frequent morbidity, determine the extent of exploitation of that genetic potential. Inadequate dietary intake and/or infections reduce nutrient availability at cellular level resulting in growth retardation. During periods of severe deprivation, linear growth rate slows down and leads to stunting. Thus stunting is a consequence of chronic food deficiency.

Technique :

A vertical measuring rod called an anthropometer is used in the older children and adults for measuring the height. In children below the age of two years who cannot stand properly, recumbent length (crown - heel length) should be measured with an infantometer.

The subject should stand erect looking straight on a leveled surface, without shoes, with heels together and toes apart. The anthropometer rod should be placed behind the subject in the centre of the heels perpendicular to the ground. The investigator standing on the side of the subject should firmly hold the chin of the subject with his left hand and the occiput of the subject with his right little finger in the 'Frankfurt Horizontal Plane' (an imaginary line joining the tragus of the ear and the eye). The moving head piece of the anthropometer should be placed in the sagital plane over the head of the subject applying a slight pressure to reduce the thickness of hair. The reading should be taken when the anthropometer rod is still in position. An average of three measurements should be taken.

MID-UPPER ARM CIRCUMFERENCE:

Poor musculature and wasting are cardinal features of protein energy malnutrition in early

childhood. Mid-upper arm circumference (MUAC) and calf circumference are recognized to indicate the status of muscle development. The mid - calf and mid-upper arm are heavily muscled and approximately circular. Of the two, the mid-upper arm is considered more feasible as it is simpler and easily accessible in any age and in both sexes.

Technique : The mid-upper arm circumference is taken on the left hand. The mid-point between the tip of the acromion and the tip of the olecranon is, located with the arm flexed at the elbow; and marked with a marker pen. The arm is left hanging freely and the fibre glass tape (not the usual tailor's plastic or cloth tape) is gently, but firmly placed embracing the arm without exerting too much pressure on the soft tissues. The reading is taken to the nearest millimeter, with the tape still in position.

SKINFOLD THICKNESS/ FAT FOLD THICKNESS:

The skinfold thickness may be measured at different sites on the trunk (sub-scapular, suprailiac, abdominal etc.) or on the extremities (triceps, thigh, mid-calf). The skinfold at triceps is more reliable than that at sub-scapular region in the assessment of obesity. The triceps skinfold is more sensitive to the socio-economic environment than the sub-scapular fatfolds.

Technique :

At the triceps :- the measurement should be made on the dorsal side at the same mid-point where mid upper arm circumference is measured. The skinfold is picked up between the thumb and the forefinger about one centimeter above the mid-point, taking care not to include the underlying muscle. It should be measured carefully, as thickness of the fat layer changes very rapidly over relatively short distances. The tips of the skinfold caliper should be applied at the mid-point at a depth equal to the skin-fold. The sub-cutaneous fat gets compressed if the caliper is kept for a longer time. The skinfold should be held gently in the left hand throughout the measurement. Average of two measurements should be taken.

At the sub-scapula :- The fat layer is fairly uniform over this site. The fatfold is measured just below and lateral to the angle of the left scapula by picking it up with the thumb and forefinger in a line running approximately 45° to the spine, in the natural line of skin cleavage. Some of the standard calipers are Harpenden, Lange and Best. Una Caliper which is available in India is also found to be reliable.

HEAD AND CHEST CIRCUMFERENCE :

Head size relates mainly to the size of brain which increases quite rapidly during infancy. The chest in a normally nourished child grows faster than head during the second and third year of life. As a result, the chest circumference overtakes head circumference by about one year age. In PEM in children, due to poor growth of chest, the head circumference may remain higher than the chest even at the age of 2 and half to 3 years. The head and chest circumferences are measured with a flexible fibre-glass tape used for measuring arm circumference. The chest circumference is taken at the nipple level preferably in mid inspiration. The head circumference is measured passing the tape round the head over the supra-orbital ridges (just above the eyes) of the frontal bone in front, and the most protruding point of the occiput on the back of the head. Slight pressure should be exerted to allow for the thickness of the hair. However, both these measurements are not very useful in routine nutritional surveys, particularly in children beyond the preschool age.

USEFULNESS OF ANTHROPOMETRIC MEASUREMENT

1. Assessment of the extent of under nutrition of vulnerable groups of population.
2. For growth monitoring of children at regular intervals (monthly) to detect at an early stage any alteration in growth during the intervals e.g. in ICDS/Under-5 clinics. This helps in early detection of failure to thrive and assists in initiating prompt remedial measures.
3. Identification of children who are at risk of under nutrition so that the nutrition programs can be specifically targeted to those groups of population.
4. For mid-term appraisal or terminal evaluation of prophylactic nutritional intervention programmes.
5. For assessing the impact of nutritional rehabilitation programs.
6. For the purpose of nutritional surveillance.
7. To assess the impact of seasonal food supply on nutritional status of the community.

COMMONLY USED ANTHROPOMETRIC CLASSIFICATIONS

Classification	Indicator	Cut-off level as % of NCHS median	Type / degree of Malnutrition	Usefulness
Gomez	Weight/age	< 60	Severe	Index of Current & Past nutritional status
		60 - 75	Moderate	
		75 - 90	Mild	
		> 90	Normal	
Indian Academy of Paediatrics	Weight/age	< 50	Grade IV	Index of Current & Past nutritional status
		50-60	Grade III	
		60- 70	Grade II	
		70-80	Grade I	
		> 80	Normal	

Classification	Indicator % W eight/Age Weight/Ht. Cut-off level as % of NCHS median			Type/degree of malnutrition	Usefulness
Waterlow	> 90	>80		Normal	
	> 90	< 80	Wasted	Short duration Malnutrition	
	< 90	> 80	Stunted	Long duration Malnutrition (nutritional dwarf)	Identifies type and duration of malnutrition
	< 90	< 80	Stunted and wasted	Current and long duration malnutrition	

III. SPOTS

1. Nutrition

Nutritional deficiency diseases being very common in our country and also being the chief predisposing factor for infectious diseases, have a great importance in Preventive and Social Medicine.

In PSM practicals the student is expected to know the nutritive value of common foods, food exchange values, the important nutrients, their rich sources and deficiency signs and symptoms. The student is also expected to know diets in certain diseases and during pregnancy and lactation. All these aspects of nutrition are given here in the form in which they are usually asked. The student is also advised to visit the kitchen in his house and take a look al the commonly used cereals pulses and millets so as to be able to identify them easily.

A. INFANT FEEDING

Breast milk is the best milk for an infant. There is no better substitute, because of its biological superiority and availability with ease. Breast feeding should be continued as long as breast milk is available but prolonged breast feeding should be avoided. Weaning at the proper time is necessary to maintain the nourishment of the child.

If breast milk is not available or is insufficient for the baby's requirement, supplementary feeds may be necessary. In case of top feeding, the sterilization of all the utensils, bowls, cups, spoons etc. or bottles and nipples should be looked into. Necessary dilution should be done with sterile water.

AGE	NATURE OF FEEDS	FREQUENCY
1 st week	Both the breasts at one sitting or half strength milk	3-4 hourly
1-4 weeks	Breast milk or 3/4 strength milk with sugar	2-3 hourly
1-3 months	Breast feeds 10-15 minutes each breast or full strength milk with sugar	3 hourly feeding schedule 3-4 ozs per feed
3-6 months	(a) Breast milk + suplementary feeds if hungry. or full strength milk 4-8 oz./feed.	3-4 hourly feeds 5-6 feeds/day
6 months to 1 year	(a) Breast milk and supplementary feeds to continue Or full strength milk 8 oz./feed (b) Second solid food	4 hourly schedule 4-5 feeds/day 2-3 times a day
1-3 years	(a) Full strength milk (b) Third solid food Breast feeds should stop by around 1 year	2-3 times a day

1. First Solid Food:

a) Cereals - rice, maize or wheat cooked with milk and sugar and with powdered pulses made into a gruel.

b) Egg yolk.

c) Mashed banana.

d) Fruit juices or soups.

2. Second Solid Food :

a) Mashed Potatoes.

b) Mashed and seived vegetables.

c) Mashed fruits like banana, chickoo, apple etc.

d) Rice and dal or khichadi, idli, etc.

3. Third Solid Food:

a) Mashed pulses.

b) Chapati or bread.

c) Vegetable and fruits.

d) Fish flour.

e) Eggs.

f) Ground meat.

For premature babies or 'small for date' babies, it is necessary that these babies be under expert guidance as they require close supervision.

Sick infant or dehydrated baby should not be starved if feeds are tolerated. An adjustment with feeds may be necessary depending upon the underlying clinical condition.

School Children :

For children it is preferable to give food at fixed times. However, the pattern depends on the school timings, but normally it can be –

At 8.00 a.m. : Breakfast

At 1 0.00 a.m. : Fruit juice, biscuit or toast etc.

At 1.00 p.m. : Lunch

At 4.00 p.m. : Afternoon snack

At 7.00 p.m. : Dinner

Bed time : Milk

Breakfast : Milk –1 cup with sugar and corn-flakes or other breakfast cereal.
Bread with butter and jam or chapati with ghee and sugar or honey.
Any one preparation of egg or cheese.

At 10.00 a.m. : Biscuits and fruit.

At 1.00 p.m. : Chapaties, rice, vegetables
Dal or fish or egg, curds.

At 4.00 p.m. : Milk – 1 cup
Biscuits or some snacks.
Roasted groundnuts and chanas.

At 7.00 p.m. : Chapaties, Rice, Dal,
Vegetable salad

At bed time : Milk – 1 cup with sugar or custard or pudding.

Diet in Pregnancy & Lactation

Early morning : Bed Tea/Coffee
- 1 cup with
1 tsp. of sugar

At 8.00 a.m. : 2 toast with butter
or 2 paratha.

At 11.00 a.m. : Fruit - one.

At 1.00 p.m. : 1 wati rice
1 wati dal
curd
leafy vegetable
fish or meat

At 4.00 p.m. : Light tea - 1 cup
Biscuits or idli
or upma or
toast.

At 6.00 p.m. : Fruit - one

At 8.00 p.m. : Khichadi
cooked vegetable
chapati or Bhakri
Pulses/root vegetable
fish or meat

At bed time : Milk – 1 cup or
custard.

PROTEINS, FATS, CARBOHYDRATES AND WATER

Nutrient	Requirement	Calorirs supplied	Utility Sourcr	
1. Protein:				
Total protein	1gm/kg/day Add 15gms for pregnancy and 25gms for lactation	4Kcal/gm	1. For repair of wear and tear, in the body	
First class protein	0.6 gm/kg		2. Production of a. Digestive juices b. Hormones c. Plasma proleins d. Enzymes e. Haemoglobins f. Vitamins Deficiency leads to kwashiorkor	First class proteins containing essential amino acids - Meat, fish, poultry, Eggs, Milk Second class proteins do not conlain all essential amino acids in adequate quantity - Pulses, Cereals, Millet,
2. Fat	1gm/kg/day (30% of total calories divided in 10% poly saturaled 10% to 20% saturated fat and less than 300mg of cholesterol)	9Kcal/gm	1. To supply calories 2. To make diet palatable 3. To help in absorption of fat soluble vitamins 4. To supply essential fatly acid 5. It produces satiety as it delays the gastric emptying 6. Concerned with the production of steroidal hormones	1. Vegetable oils like groundnut oil. coconut oil, mustard oil. 2. Milk. 3. Meat, fish, yellow of an egg. Commercial preparation is portagen which contains skimmed milk, sucrose, corn starch, coconut oil and saftlower oil.
3. CHO carbohydrates	200 to 300gms/day (60 to 70% total calories)	4Kcal/gm	1. To supply immediate calories 2. To prevent hepatic damage 3. It has protein sparing effects	1. Rice. wheat, bajra and pulses. 2. Fruits. vegetables and tubers. 3. Sugar, honey, jaggery. 4. Milk
4. Water	1ml/Kcal Increased requirement occurs in - a. Diarrihoea b. Vomiting c. Burns d. Excessive diaphoresis		1. Regulation of body temperature 2. Correction of dehydration 3. Maintaining, the texture of skin 4. Production of digestive juices and enzymes.	1. Drinking water 2. Beverages 3. By-producls of food metabolism.
5. Calories	For an adult 30 cal/kgsedentary worker 35 cal/kg moderate worker 40 cal/kg heavy worker It depends upon age, sex, occupation and climate. 300 calories should be added for pregnancy. 600 calories should be added for laclation. 300-500 cat should be reduced for obesity.	For ATP production and various cellular functions	Deficiency leads to Marasmus.	

VITAMINS AND MINERALS

Nutrient	Requirement Male	Requirement Female	Deficiency Symptoms, and Signs and tioxicity	Sources
1. Vitamin A	5000 IU/Day	4000 IU/day + 200 in Pregnancy + 400 in Lactation	1. Follicular hyperkeratotis. 2. Night blindness. 3. Xerophthalmia and keratomalacia. 4. Bitot's spots. 5. Hyposmia. **Toxicity** 1. Abdominal pain. 2. Nausea, vomiting. 3. Headache. 4. Dizziness. 5. Benign intracranial hypertension.	Whole milk, egg, liver and fish liver oil, butter, fruits like papaya, ripe mango, orange. Dark green leafy vegetable, carrots, drumstick leaves.
2. Vitamin D	400 IU/day	400 IU/day +200 in Pregnancy +200 in Lactation	1. Tetany. 2. Rickets. 3. Osteomalacia. **Toxicity** 1. Anorexia, nausea, vomiting. 2. Hypercalcemia. 3. Renal stones. 4. Renal failure. 5. Metastatic calcification.	Milk, butter, fish liver oil, egg yolk and liver.
3. Vitamin E	30mg/day or 12-15 IU/day	25-30mg/day or 10-12 IU/day +5mg in pregnancy +5mg in Lactation.	1. Anemia. 2. Nocturnal muscle cram 3. Infertility. Toxicity: Not known.	Germinating cereals, wheat, lettuce, maize, peas, whole rice, meat.
4. Vitamin K	Exat requirement not known. approx. 70-140 μg/day		Hemorrhagic diathesis like petechiae, Purpura, Echymosis	Green vegetables, alfalfa, spinach, cabbage, egg yolk, tomatoes, vitamin K_1 synthesised by bacteria in intestine.

VITAMINS AND MINERALS

Nutrient	Requirement		Deficiency Symptoms, and Signs	Sources
	Male	Female		
5. B_1 (Thiamine)	1-2mg/day	1-1.5 mg/day +0.5mg in Pregnancy. +0.5mg in Lactation.	1. Dry beriberi-peripheral neuropathy Wernicke encephalopathy, korsakoff psychosis, muscle tenderness, weakness, hyporeflexia. 2. Wet beriberi-Tachycardia, cardiomegaly, congestive heart failure.	Dried yeast, wheat germ, whole cereals, leg urnes, soya beans, peanut, cashewnut, liver.
6. B_1 (Riboflavin)	1 to 2mg	1 to 1.5 mg 0.5mg in Pregnancy. 0.5mg in Lactation	Angular cheliosis, stomatitis (magenta coloured tongue), corneal vascularisation, scrotal dermatosis, angular blepharitis.	Milk, eggs, liver, germinating seeds, leafy vegetable.
7. B_1 (Nicotinic acid or PP factors).	20mg	15-20mg +2 in Pregnancy. +5 in Lactation	Dermatitis (erythema, pigmentation). Diarrhoea, Dementia, red and raw tongue. atrophic lingual papillae, fissuring of tongue.	Enriched bread, whole cereals, meat, fish liver.
8. B_6 (Pyridoxine)	2mg	1.5mg 0.5 in Pregnancy. 0.5 in Lactation.	Nasolabial seborrhea, glossitis, oxalate renal stones, peripheral neuropathy.	Yeast, liver, meat, cereals, milk, eggs, spinach, leafy vegetables.
9. Folic acid	100μgm	100μgm +20 in Pregnancy. +20in Lactation.	Megaloblastic anaemia, glossitis, stomatitis, diarrhoea.	Meat, liver, egg, green leafy vegetables, legumes.
10. Biotin	300mg	300mg. +20 in Pregnancy. +40 in Lactation.	Fatigue, muscle pain, depression, dermatitis.	Liver, eggs, meat.
11. Pantothenic acid	10mg	5 to 10mg +1 in Pregnancy. + 1 in Lactation.	Burning feet syndrome, sleep disturbances.	Liver, kidney, eggs, whole grain and milk.
12. Cyanocobalamin (B_{12})	1mg	1 to 2mg +1mg in Pregnancy. +1mg in Lactation.	1. Megaloblastic anaemia. 2. Peripheral neuropathy. 3. Optic neuritis. 4. Subacute combined degeneration of the spinal cord. 5. Mental changes.	Liver, meat, fish, egg, milk, kidney.

VITAMINS AND MINERALS

Nutrient	Requirement		Deficiency Symptoms, and Signs	Sources
	Male	Female		
13. Ascorbic acid (Vit C)	50mg	50mg +20 in Pregnancy. +40 in Lactation.	1. Scurvy. 2. Petechiae, echymosis, bleeding gums. 3. Delayed wound healing. 4. Microcytic normochromic anaemia.	Amla, citrus fruits, sweet lime, guava, mango, orange, pineapple, leafy, vegetable, milk, fish, egg yolk.
14. Sodium	2-3m Eq/kg	2-3m Eq/kg	Symptoms and signs of hyponatremia are- 1. Muscular twitches. 2. Irritability. 3. Convulsion. 4. Altered sensorium. 5. Coma.	1. Table salt. 2. Salad, papad. 3. Salty biscuits, canned food. 4. Meat, fish. 5. Instant cooked cereals. 6. Butter milk. 7. Green mango, lichies, jack fruit.
15. Potassium	1-2m Eq/kg	I-2m Eq/kg	Hypokalemia: 1. Neuromuscular disturbances like muscle weakness, paraesthesia, hyporeflexia. 2. Cardiovascular arrhythmia, increased digitalis sensitivity, orthostatic hypotension. 3. Nephropathy. 4. Metabolic alkalosis. 5. Worsening of hepatic encephalopathy.	1. Fruits and fruit juices. 2. Coconut water. 3. Milk. 4. Meat, fish. 5. Potato, sweet potato, tomatoes. 6. Cold drinks. 7. Pulses.
16. Calcium	400-500mg/day	400 to 500mg/day +400 in Pregnancy. +400 in Lactation.	1. Peri-oral and peripheral paraesthesiae. 2. Carpopedal spasm. 3. Bronchospasm and laryngospasm. 4. Convulsion. 5. Chvostek's sign. 6. Trousseau's sign. 7. Prolonged QT interval in ECG.	Milk and milk products, fish, crabs, ragi, horse gra, soyabean, tumip, chuna (slaked lime) with betel leaves, poppy seeds, coconut, sago, custard apple.
17. Phosphorus	800-1000mg/day	800 to 1000mg/day +400 in Pregnancy. +400 in Lactation.	1. Anorexia. 2. Dizziness. 3. Bone pain and osteomalacia. 4. Hemolytic anaemia.	Milk and milk products, cheese, egg, fish, meat, cereals, pulses, nuts.

VITAMINS AND MINERAlS

Nutrient	Requirement		Deficiency Symptoms, and Signs	Sources
	Male	Female		
18. Iron	25mg/day	35mg/day. +5mg in Pregnancy.	1. Hypochromic microcytic anemia. 2. Required in the body for the formation of Haemoglobin. Myoglobin. Catalases. Cytochromes. Peroxidases.	1. Liver, meat, fish. 2. Cereals, pulses, green leafy vegetables. 3. Nuts, oilseeds. 4. Jaggery. 5. Dryfruits, raisins, dates, apricots. 6. Iron/folic acid tablets given as part of anemia control programme. 7. Iron fortified salt.
19. Iodine	0.15mg.	0.10mg. 0.25mg in Pregnancy and Lactation.	1. Hypothyroidism. 2. Goitre 3. Mental retardation, Cretinism 4. Reduced BMR. 5. Obesity 6. Slow and sluggish behaviour. 7. Low voltage ECG.	1. Sea food. 2. Salt. 3. Water. 4. Cereals. 5. Spinach. Note that cabbage, cauliflower and radish act as goitrogens by interfering with the absorption of Iodine in diets.
20. Fluorine.	1.5 to 4 mg/day	1.5 to 4mg per day.	(0.5 to 0.8mg/litre of drinking water) less than 0.5mg/litre is associated with Dental caries. While more than 1.2mg/litre is associated with i) Dental fluorosis. 1. Mottled enamel. 2. Brown, Black teeth. 3. Corrosion, pitting of teeth. ii) Skeletal fluorosis. 1. Pain and stiffness of joints. 2. Exostosis and entrapment neuropathy 3. calcification of tendons, ligaments and interosseous membranes. 4. Osteoporosis. 5. Genu Valgum and Bamboo spine deformity.	1. Sea food. 2. Salt. 3. Water. 4. Cheese. 5. Tea.

Cereal Exchanges equivalent to 100 calories

		Approximate weight
Bread	2 slices or 1 small loaf	40 gms
Or Fulka	2 small, thin	15 gms flour for each
Or Chapati	1 medium size	30 gms flour without fat
Or Paratha	1 small with 1 tsp. fat	20 gms flour with 1 tsp. fat
Or Bhakari	1 small or 1/2 medium	30 gms flour
Bhakari made from jowar, ragi, bajara or rice or wheat.	size, medium thick	
Or Cooked rice	1 medium size bowl	30 gms raw
Or Rice flakes	1 medium bowl	30 gms
Or Suji .	1 handful	30 gms

Dal and Pulses exchange equivalent to 100 calories

Thick dal	1. medium bowl	30 gms raw
Or Thin dal	2 medium bowls	30 gms raw
Or Semisolid dal	1½ medium bowl	30 gms raw
Or pulses usal	3/4 medium bowl, semisolid'	30 gms raw

30 gms dal or pulse is roughly equal to a handful of raw dal of a housewife.

Milk exchange equivalent to 120 calories

Buffalo's milk	1 small cup	100 ml.
Toned milk (3% fat)	1 glass	200 ml.
Standard milk (5% fat)	3/4 glass or 1 cup	150 ml.
Skimmed milk.	2 glasses or approximately 3 cups	400 mi.
Curds (4% fat)	1½ medium bowl	200 gms.

Meat fish and egg equivalent to 100 calories

Meat (average)	5-6 small pieces	75 gms.
Fish	3 medium size pieces	90 gms.
Egg	1½ large size	70 gms.
Chicken	3-4 medium size pieces or 1/8 of a medium size bird.	90 gms.

Alcohol

Bear	240 mI. (1 glass)	98 calories.
Brandy	10 z.	73 calories.
Gin	43 mI. (1 measure)	105 calories.
Rum	43 mI. (1 measure)	105 calories.
Sherry	43 mI. (1 measure)	120 calories.
Scotch whisky	43 mI. (1 measure)	119 calories.

Fruit exchange. Approximately 50 calories

1.	Apple	2 "diameter	80 gms
2.	Banana	1 small or 1/2 big	50 gms
3.	Chikku	1 medium size	75 gms
4.	Dates	2	15 gms
5.	Grapes	10 to 12	80 gms
6.	Mango	1 small or 1/2 big size	10 gms
7.	Orange	1 medium	120 gms
8.	Papaya	1/3 medium fruit	150 gms
9.	Pineapple	1/2 cup cubed or 2-3 slices	80 gms
10.	Raisins	2 tb. spoon leveled	15 gms
11.	Red tomato	1 big	125 gms
12.	Sitaphal	1/4 big or 1/2 medium	40 gms
13.	Sweet lime	1 medium (Mosambi)	140 gms
14.	Water melon	2 - 3 slices	250 - 300 gms
15.	Coconut water	1 glass	200 ml

Values per 100 gms Edible Portion

Foods	Proteins	Fats	Carbo hydrates	Calories	Calcium (mg)	Iron (mg)	Minerals/ Vitamins
					important source only		
Cereals :							
Bajra	11.6	5.0	67.5	361	42	8.0	Vit A (132 μg carotene)
Cholam (Jowar)	10.4	1.9	72.6	349	-	4.1	-
Maize, dry	11.1	3.6	66.2	342	-	-	-
Ragi	7.3	1.3	72.0	328	344	3.9	-
Rice, raw, milled	6.8	0.5	78.2	345	-	-	-
Rice, Flakes	6.6	1.2	77.3	346	20	20.0	Folic add 142 μ g
Rice, puffed	17.5	0.1	73.6	325	23	6.6	-
Sujj (Rawa)	10.4	0.8	74.8	348	-	-	-
Wheat, whole	11.8	1.5	71.2	346	-	5.3	-
Wheat flour (atta)	12.1	1. 7	69.4	341	-	4.9	-
Pulses and Legumes:							
Bengal gram, whole	17.1	5.3	60.9	360	202	4.6	Vit A189 μ g carotene
Bengal gram,dal	20.8	5.6	59.8	372	56	5.3	Vit A 129 μ g carotene
Chavli, small	24.6	0.7	57.1	333	77	8.6	-
Chavli big	23.9	1.3	57.2	336	"	"	-
Green gram, whole	24.0	1.3	56.7	334	124	4.4	-
Green gram, dal	24.5	1.2	59.9	348	75	3.9	-
Rajmah	22.9	1.3	60.6	346	260	5.1	-
Red gram dal (Turdal)	22.3	1.7	57.6	335	73	-	-
Soya bean	43.2	19.5	20.9	432	240	10.4	Vit A 426 μ g carotene

Foods	Proteins	Fats	Carbo hydrates	Calories	Calcium (mg)	Iron (mg)	Minerals/ Vitamins
					important source only		
Leafy vegetables :							
Amaranth tender (Math)	4.0	0.5	6.3	46	397	3.45	Vit.A (5500 lg carotene) Vit.C = 100 gms.
Ambat chuka	1.6	0.3	1.4	15	63	-	VitA 3660 μg
carotene Cabbage	1.8	0.1	4.6	27	-	-	VitC 124 mg.
Caulinower greens	5.9	1.3	7.9	66	340	8.8	VitC 56 mg.
Drumstick leaves	6.7	1.7	12.5	92	440	-	Vit.A (6780 μg carotene) Vit.C (220 mg)
Fenugreek (Methi)	3.5	0.9	6.0	49	395	1.93	Vit.A (2340 μg carotene)
Root vegetables :							
Beet root	1.7	0.1	8.8	43	-	-	-
Carrots	0.9	0.2	10.6	48	80	-	Vit.A (1890 ug carotene)
Onion	1.8	0.1	12.6	59	47	-	-
Potato	1.6	0.1	22.6	97	-	-	-
Radish, white	0.7	0.1	3.4	17	35	-	-
Radish, pink	0.6	0.3	6.8	32	50	-	-
Sweet potato	1.2	0.3	28.2	120	46	-	-
Tapioca	0.7	0.2	38.1	157	50	-	-
Other vegetables :							
Bitter Gourd	1.6	0.2	4.2	25	-	-	(Vit.C 100 mg)
Brinjal	1.4	0.3	4.0	24	-	-	-
Calabash cucumbcr Dudhi)	0.2	0.1.	2.5	1.2	-	-	-
Cucumber	0.4	0.1	2.5	13	-	-	-
Drumstick (pulp only)	2.5	0.1	3.7	26	30	-	(Vit.C 120 mg)
Green papaya	0.9	0.1	6.2	29	-	-	-
Lady finger (Bhendi)	1.9	0.2	6.4	35	66	-	-
Mango green	0.7	0.1	10.1	44	-	-	-
Pumpkin red	1.4	0.1	4.6	25	-	-	-
Ridge Gourd	0.5	0.1	3.4	17	-	-	-
Snake Gourd (Padwal)	0.5	0.3	3.3	18	-	-	-
Tinda tender	1.4	0.2	3.4	21	-	-	-
Tomato green	1.9	0.1	3.6	23	-	-	(Vit.A 192 μg) Carotcn
Nuts and oil seeds:							
Almond	20.8	58.9	10.5	655	230	5	-
Cashewnut	21.2	46.9	22.3	596	50	6	-
Coconut, dry	6.8	62.3	18.4	662	400	8	-
Groundnut	26.7	40.1	20.3	549	90	-	-
Pistachionut	19.8	53.5	16.2	626	140	8	-
Walnut	15.6	64.5	11.0	687	100	3	-

Foods	Proteins	Fats	Carbo hydrates	Calories	Calcium (mg)	Iron (mg)	Minerals/ Vitamins
					important source only		
Fruits :							
Amla	0.5	0.1	13.7	58	50	-	(Vit.C 600 mg)
Apple	0.3	0.1	13.3	55	-	-	-
Banana (green)	0.8	0.8	24.4	108	-	-	-
Dates (dried)	2.5	0.4	75.8	317	120	7.3	-
Jack fruit	1.9	0.1	19.8	88	-	-	-
Lemon (Limhu)	1.0	0.9	11.1	57	70	-	(Vit.C 40 mg)
Lime (Mosambi)	1.5	1.0	10.9	59	40	-	(Vit.C 50 mg)
Mango,	0.5	0.8	18.2	82	-	-	(Vit.A 2743 μg carotene)
Orange	0.6	0.2	8.9	40	-	-	(Vit.A 1104 μg carotene) (Vit.C 30 mg)
Papaya (Ripe)	0.6	0.1	7.2	32	-	-	Vit.A 666 μg carotene Vit.C 57 mg
Pincapple	0.4	0.1	10.8	46	-	-	-
Pomegranate (Dalimb)	1.6	0.6	14.5	65	-	-	-
Sapota (Chikku)	1.1	1.2	25.8	118	28	-	-
Silaphal	1.6	0.3	26.2	114	17	4.31	-
Tomato (Ripe)	0.9	0.2	3.6	20	48	-	Vit.A 351 μg carotene Vit.C 27 mg
Fishes and Marine Products:							
Black pomfret	20.3	2.6	1.5	111	286	2.3	-
Crab (small)	11.2	9.8	9.1	169	1606	-	Niacin 3.1 mg
Lobster	20.5	0.9	-	90	-	-	-
Pomfret (While)	17.0	1.3	1.8	87	200	-	-
Prawn (Muscle)	20.8	0.3	-	86	323	5.3	Niacin 4.8 mg
Rohu	16.6	1.4	4.4	97	650	1	Cholinc= 819 mg.
Sardine	21.0	1.9	-	101	90	-	-
Shark	22.9	0.7	-	98	357	-	-
Bombay Duck (dry)	61.7	4.0	2.5	293	1389	19	-
Shrimps, small (Dried)	68.1	8.5	-	349	4384	-	-
Other Flesh Foods :							
Beef (Muscle)	22.6	2.6	-	114	68	19	-
Duck	21.6	4.8	0.1	130	-	-	-
Egg (Duck)	13.5	13.7	0.8	181	70	2.5	Vit.A 360μg
Egg (Hen)	13.3	13.3	-	173	60	2.1	Vit.A 360μg
Fowl	25.9	0.6	-	109	-	-	-
Goat meat muscle	21.4	3.6	-	118	-	-	-
Liver (Goat)	20.0	3.0	-	107	-	-	-
Liver (Sheep)	19.3	7.5	1.3	150	-	6.3	Vit.A 690 μg
Mutton (Muscle)	18.5	13.3	-	194	150	2.5	-

Foods	Proteins	Fats	Carbo hydrates	Calories	Calcium (mg)	Iron (mg)	Minerals/ Vitamins
					important source only		
Fats and Edible Oils:							
Butter	-	81.0	-	729	-	-	Vit.A 3200μg carotene
Ghee	-	100.0	-	900	-	-	Vit.A 2000 μg carotene
Vegetable cooking oil	-	100.0	-	900	-	-	-
Vanaspati	-	100.0	-	900	-	-	-
Milk and Milk Products:							
Milk, cow's	3.2	4.1	4.4	67	120	-	Vit.A 174 μg
Milk, buffalo's	4.3	8.8	5.1	117	210	0.2	Vit.A160 μg
Milk, goat's	3.3	4.5	4.6	72	170	0.2	Vit.A 182 μg
Milk, human	1.1	3.4	7.4	65	28	-	Vit.A 137 μg
Curds	3.1	4.0	3.0	60	149	-	-
Cheese	24.1	25.1	6.3	348	790	-	Vit.A 273 μg
Skimmed milk powder (cow's milk)	38.0	0.1	51.0	357	1370	-	-
Whole milk powdcr (cow's milk)	25.8	26.7	38.0	496	950	-	Vit.A 1400 μg carotene
Standardized milk	4.1	5.0	4.5	80	200	-	-
Miscellaneous - Foodstuffs:							
Arrowroot flour	0.2	0.1	83.1	334	-	-	-
Bread, white	7.8	0.7	51.9	245	-	-	-
Bread, brown	8.8	1.4	49.0	244	-	-	-
Cane sugar	-	-	99.4	398			
Coconut (Tender)	0.9	1.4	6.3	41	-	-	-
Honcy	0.3	-	79.5	31.9			
Jaggery cane	0.4	0.1	95.0	383	80	2.64	-
Neera	0.4	-	10.9	45	-	-	-
Papad	18.8	0.3	52.4	288	-	-	-
Sago	0.2	0.2	87.1.	351	-	-	-
Sugar cane juice	0.1	0.2	9.1	39	-	-	-
Toddy (Sweet)	0.1	0.2	14.3	59	150	0.3	-
Toddy (Fermented)	0.1	0.3	1.8	10	-	-	-

Household measures :

1	tsp.	=	1teaspoon	=	5 ml.
1	tb. spoon	=	1 table spoon	=	15 ml.
1	Bowl	=	150 ml.		
1	cup	=	150 ml.		
1	glass	=	200 ml		

B. DIET IN DISEASE

1. Kwashiorkor and Marasmus

a. Proteins: 20% of the total calories should be supplied by the proteins. This amounts to 3 to 3.5 gms per kg of the expected body weight or approximately 45 to 55 gms of proteins per day. Adequate supply of proteins is necessary to prevent and treat Kwashiorkor. A good source of proteins is milk. Skimmed milk and Bengal gram (chana) are rich in proteins and may also be used. Latter is relatively inexpensive and may be used for poor patients.

b. Fats: Fats make the diet palatable and are supplied to make up 15-20% of the total calories.

c. Carbohydrates: To supply the remaining required calories.

d. Calories: The daily requirement for the child is about 90-100 calories per kg of expected body weight. For a child of 2 years, the requirement is about 1000 to 1200 calories per day.

e. Vitamins: Multivitamin preparations are helpful as vitamin deficiencies are also associated with gross malnutrition.

f. Minerals: Serum potassium level is invariably markedly low, hence oral administration of potassium salts is advisable. Calcium supplements such as calcium lactate is useful. Iron therapy is advised after a week of high protein diet, otherwise with a low serum transferrin, it may increase free circulating iron, which may result in hemosiderosis. Use of magnesium sulphate heptahydrate 50% solution improves ECG along with clinical improvement.

g. Miscellaneous: Parenteral therapy can be dangerous as the fluid and electrolyte disturbances may be casily precipitated. There fore it is best to administer all nutrients orally.

DIET SHEET

Early morning	: Milk – 1 cup with sugar 1 tsp.
Breakfast	: Bread – 1 slice. Egg- one.
At 10.00 a.m.	: Banana – one.
At 12.00 noon	: Dal dhokali – 3/4 medium bowl Rice – 1/2 medium bowl. Curds – 1/2 medium bowl.
At 2.00 p.m.	: Golpapadi – 30 gms – 2 pieces.
At 4.00 p.m.	: Boiled moong or other pulses, salad with carrots, tomato, boiled potato and onion – 1 medium bowl.
At 6.00 p.m.	: Milk - 1 cup with sugar 1 tsp.
At 7.00 p.m.	: Rice -1/2 medium bowl, Dal-1/2 medium bowl. Thepla with leafy vegetable – one small bowl.
Bed time	: Milk - 1 cup with sugar 1 tsp.

The diet provides 1200 calories with 50 gms proteins, 37 gms fats and 171 gms carbohyrates.

2. Cirrhosis of liver:

Conscious patient without jaundice (compensated cirrhotics)

Principle:

1. Proteins: High protein diet is required as it prevents
   a. Fatty changes (because of choline)
   b. Hepatic necrosis (because of cystine)
   c. Cirrhosis of liver (because of methionine)

   It is necessary for regeneration of liver cells.
2. Fats: 1 gm/kg is allowed.
3. Carbohydrates:Large quantities are advocated because.
   a. They protect the liver cells against the damage from the injurious agents.
   b. They prevent the endogenous protein breakdown by their protein sparing effect.
   c. They provide large quantities of calories.
   d. Liver function improves when there is an adequate intra-hepatic glycogen store.
4. Calories: High calorie diet is recomended patient is usually undernourished.
5. Vitamins: Vitamin B-complex has a beneficial effect on liver. B_{12} is lipotropic and probably aids in transmethylation with a possible choline sparing effect.
6. Minerals: Sodium is to be restricted if there is retention of fuids and potassium supple ments are necessary with diuretic therapy.
7. Alcohol should be avoided because:
   a. It is hepatotoxic.
   b. It increases the need for proteins and vitamin B- complex.
   c. It produces gastritis which produces anorexia and vomiting leading to malnutrition.

DIET SHEET

Early Morning : Light tea -1 cup with sugar 2 tsp. and milk 25 ml.

Breakfast : Skimmed milk -1 cup with sugar 1 tsp.
Bread - 2 slices with jam 1. table spoon.

At 10.00 a.m. : Fruit - one banana or fruit juice 1 cup with sugar 1 tsp.

At.12.00 Noon : Rice -1 medium bowl
Fulka - 4 small, thin, or Chapaties – 2 medium size.) with ghee ltsp.
Dal- 1/2 medium bowl thin dal
Egg - One (half boiled); or
Fish - 2 medium size pieces with curry.
Cooked pumpkin - 3/4 medium bowl
Curds - 1/2 medium bowl with sugar 1tsp.
Oil for cooking – 2 tsp.

At 3.00 p.m. : Skimmed milk–1 cup
with protein supplement–1 table spoon.
Pohe –1 handful with jaggery 10 to 15gms.

At 6.00 p.m. : Fruit, anyone or fruit juice 1 cup with sugar 1 tsp.

At 8.00 p.m. : Khichadi – 2 bowls.
Fulka – 2 small, or
Chapati –1 medium size.
Cooked potato vegetable – 3/4 mediurm bowl,
Boiled carrots and beet root salad 3/4 mediurm bowl,
Curd Kadhi – 1 medium bowl
Oil for cooking – 2 tsp.

Bed time : Skimmed milk – 1 cup with sugar.
Protein supplement–1 table spoon.

The diet provides 2015 calories, 72 gms proteins, 40 gms fats and 342 gms carbohydrates.

3. Obesity.

Principle :

1. Proteins : 1 gm/kg is required.
2. Fats : They should be restricted as they are concentrated source of energy. Vegetable oils contain essential fatty oils and hence are permitted as per requirement.
3. Carbohydrates: Substances rich in carbohydrates like potatoes, sweet potatoes and rice are restricted where as bulk substances which produce a sense of satiety and regulate bowel movements likc green vegelables and fruits are not restricted.
4. Calories : Low calorie diet reduces weight. In severe cases rigid restriction amounting to 600 calories is recommended.
5. Vitamins : Fat and water soluble vitamin supplements are necessary.
6. Miscellaneous:
   a. Liberal water inlake before food may help to reduce the intake of food.
   b. Food and fruits in between regular lunch and dinner should be forbidden.
   c. Large amount of chocolates and peppermints are forbidden.
   d. Exercise along with food will help to burn away excessive fat.

DIET SHEET

Early Morning: Light tea – 1cup without sugar, milk 2 tb. spoon.

Breakfast : Milk – 1cup (150 ml) without cream and sugar.
Bread – 2 slices without butter.

At 10.00 a.m. : Papaya – 2 - 3 slices.

At 1.00 p.m. : Fulka – 3 small size, thin (around 12-15 gms nourin each)
Or fulka – 2 small size, thin and rice – 1/2 medium bowl.
Or paratha – 1 medium size.
Or jowar or bajra bhakari– 1 small size.
Or rice – 1 medium bowl.
Dal–3/4medium bowl if semi-solid or 1 bowl thin dal.
Or Fish – lmedium size piece
Or 1 lean meat - 2 - 3 small pieces.

Spinach vegetable –1 medium bowl
Cucumber and onion salad – 1 medium bowl
Thin buttermilk – 1 glass
Oil for cooking – 2tsp. only

Afternoon : Light tea -1 cup with sugar and milk – 2 tb. spoon.
Bread – 1 slice or one idli, or upma - 1/2 bowl.

Dinner : Fulka – 3 small size, thin
Pulses, usal– 1/2 medium bowl
French bean vegetable – 1 medium bowl.
Cabbage salad – 1 medium bowl
Thin butter milk – 1 glass.

Bed time : Milk – 1cup (150 ml.) without sugar and cream.

The diet provides 1,200 calories, 50 gms proteins, 38 gms fats and 162 gms carbohydrates.

4. Diabetes Mellitus (DM)

1. To reduce and maintain the weight at 5% less than the calculated weight for age, sex and height.
2. Sweets are to be avoided. Instead of sugar, artificial sweetening agent like saccharine advocated.
3. Undue starvation is forbidden it might precipitate ketosis which is hazardous.

Principle :

1. Proteins : As per normal recommendation.
2. Fats : In an obese patient severe fat restriction isnccessary.
3. Carbohydrates : Carbohydrate intake must be minimized in order to reduce blood sugar. Drastic reduction of Carbohydrates is forbidden as it might result in preferential excessive metabolism of fat resulting in kctoacidosis.
4. Calories : Total calories should be adequate for the growing children and underweight persons. However, in an obese patient it might be necessary to reduce calories.
5. Minerals: Adequate amount should be supplied.
6. Vitamins : Vitamin B complex group should be given to prevent and treat polyneuritis accompanying diabetes.
7. Miscellaneous : The following food articles should he avoided
   a. Sweet drinks and carbonated drinks
   b. Cakes, pastries, cream, beer and wines
   c. Dried and canned fruits, sweet pickles, jaggery and sweetmeats.

Diet Sheet for DM

1000 calories diabetic diet for an obese diabetic :

Early Morning : Light tea – 1 cup without sugar, milk 1/4 cup.

: Milk (toned) 1 small cup without sugar.
Bread – 2 slices without butter.
Fruit – one (orange or guava or sweet lime).

Lunch : Fulka – 2 small or chapati – 1 medium size.
Dal–l/2 medium bowl semisolid
Or 3/4 to 1 bowl liquid or thin dal.
Or fish – 1 piece medium size with curry 1/2 bowl.
Or lean meat – 2 small pieces
Leafy vegetable – 1 medium bowl.
Cucumber or cabbage. Salad – 1 medium bowl,
Curds – 1/2 medium bowl.
Thin buttermilk – 1 glass.
Oil for cooking – 11/2 tsp.

Afternoon : Light tea – 1 cup without sugar
Bread – 1 slice or biscuits – 2.

Evening : Fruit – one (a small apple or 2 slices of papaya or pineapple).

Dinner : Fulka – 2 small or chapati – 1 medium size
Or rice – 1 medium bowl or bhakari – 1 small
Dal – 3/4 bowl or fish or meat as above.
Other vegetable – 1 medium bowl.
Thin buttermilk – 1 glass.
Oil for cooking – 1 1/2 tsp.

Bed time : Milk (toned) – 1 small cup (4 ozs) without sugar.

The diet provides 1,000 calories, 40 gms proteins, 33 gms fats and 135 gms carbohydrates.

1500 calories diabetic diet for a normal weight diabetic:

Early Morning: Light tea – 1 cup without sugar.
Breakfast : Milk (toned) 1 glass without sugar.
Bread – 2 slices.
Fruit – one.
Lunch : Fulka – 2 small or chapati one medium size.
Rice – 1 medium bowl.
Dal 1/2 medium bowl semisolid or 1 medium bowl thin dal.
Or fish – 2 pieces medium size with curry
Or lean meat – 2 - 3 small pieces 1/2 bowl.
Salad – 1 medium bowl.
Curds – 1 medium bowl.
Buttermilk – 1 glass.
Afternoon : Light tea – 1 cup without sugar.
Upma or pohe-1/2medium bowl with oil for cooking 1 tsp.
(Or bread - 2 slices).
Evening : Fruit– one.
Dinner : Fulka – 2 small or
bhakari – 1 small.
Rice – 1 medium bowl or 2 more fulka
Dal – 1/2 medium bowl
Vegetables –1 medium bowl.
Salad – 1 medium bowl
Buttermilk – 1 glass
Oil for cooking –1½ tsp.
Bed time : Milk (toned) 1 glass without sugar.

The diet provides 1,500 calories, 57 gms proteins, 40 gms fats and 231 gms carbohydrates.

2000 calories diabetic diet for an underweight diabetic:

Early Morning : Light tea –1 cup with sugar 1 tsp.
Breakfast : Milk (toned) 1 glass without sugar.
Bread – 4 slices with butter 1 tsp.
Fruit – one.
Lunch : Fulka – 4 small or chapati – 2 medium size.
Rice – 1 medium bowl
Dal – 3/4 medium bowl semisolid
Or fish – 2 pieces medium size } with curry
Or lean meat – 2-3 small pieces } bowl.
Leafy vegetables– 1medium bowl
Salad – 1 medium bowl
Curds – 1 medium bowl
Buttermilk – 1 glass.
Oil for cooking – 2 tsp.
Afternoon : Light tea – 1 cup with sugar 1 tsp.
Upma or pohe - 3/4 medium bowl with oil for cooking 1 tsp.
Or bread – 2 slices with butter 1 tsp.
Evening : Fruit – one.
Dinner : Fulka – 4 small or
bhakari – 2 small.
Riee – 1 medium bowl or 2 more fulka.
Dal – 1 medium bowl.
Vegetables –1 medium bowl.
Salad – 1 medium bowl
Buttermilk – 1 glass
Oil for cooking – 2 tsp.
Bed time : Milk (toned) – 1 glass without sugar.

Diet provides 2,000 calories, 72 gms proteins, 57 gms fats, and 310 gms carbohydrates.

5. Ischaemic heart disease. (IHD)

Principle:

1. Proteins: Normal intake is allowed
2. Fats: Low cholesterol and Low triglyceride Following fatty diet should be avoided:
   a. Animal fats - beaf, meat, pork and organ meat.
   b. Dairy products containing, butter, Ghee and egg yolk.
   c. Hydrogenated vegetable oils like vanaspati ghee.
3. Carbohydrates: Normal intake is taken.
4. Calories: Reduction is advised in obese patient.
5. Vitamins and Minerals as per normal requirement.

DIET SHEET

Early Morning : Light tea – 1 cup without sugar and milk 1/4 cup.

At 8.00 a.m. : Milk – 1 cup without sugar and cream.
Bread – 2 slices without buter.

At 11.00 a.m. : Chapaties – 2 medium size } With out fat
Or fulka – 4 small size
Rice – 1 medium bowl
Dal – 1 medium bowl.
Or pulses – I medium bowl.
Or fish – 2 medium size pieces } With curry 1/2 bowl
Or Chicken – 2 medium size pieces
Cooked vegetables – 3/4 medium bowl.
Salad – as desired.
Curds – 3/4 medium bowl.

At 3.00 p.m. : Light tea – 1 cup without sugar and Milk 1/4 cup.
Idlies – 2 or dhokala – 3 to 4 pieces.

At 6.00 p.m. : Any one fruit such as orange or sweet lime or papaya.

At 8.00 p.m. : Jowar bhakari – 2 medium size
Pulses usal – 3/4 medium bowl.
Cooked vcgetables – 3/4 medium bowl.
Salad – as desired.
Curds – 3/4 medium bowl.
Oil for cooking – 1 1/2 tsp. only.

At Bed time : Milk – 1 cup without sugar and cream.

6. Hypertension

Principle :

1. Proteins: 50 to 60 gms of proteins are required. It is difficult to achieve salt restriction without protein restriction.
2. Fats: High intake of animal fats and hydrogenated oils should be discouraged, as they are responsible or atheroselerosis.
3. Carbohydrates: It should constitute the major bulk of calories required for daily activities.
4. Calories: Obese patients should reduce the weight to achieve ideal weight.
5. Vitamins: As usual, supplements are recommended.
6. Minerals: Sodium must be restricted in majority of the hypertensives because it has a water retaining property, which aggravates the hypertension.
7. Water : It should be restricted i.e. Urine out put plus 500 ml is given.

DIET SHEET

Early morning : Light tea, 1 cup with sugar 1 tsp. and milk 1/4 cup.

Breakfast : Milk 1 cup without sugar and cream
Jowar roti – 2 small, thin.

At 10.00 a.m. : Coconut water – 1 glass (200 ml)

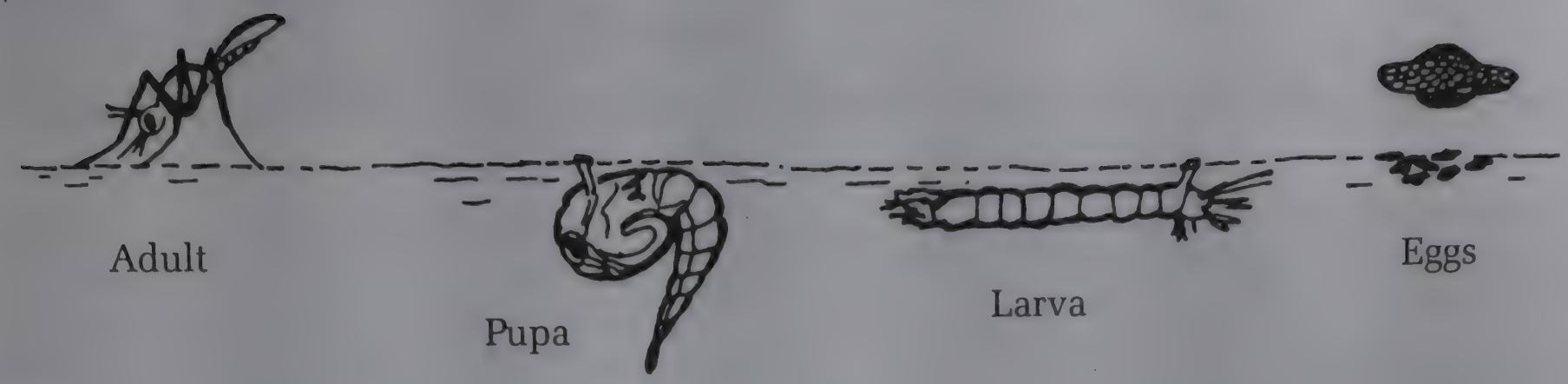
At 1.2.00 Noon : Fulka – 4 small, thin
Rice – 1 medium bowl
Dal – 1 medium bowl, or
Lean meat 4 - 5 small pieces with curry 1/2 bowl
Turai vegetable – 3/4 medium bowl.
Cucumber and onion salad – 1 medium bowl
Curds – 3/4 medium bowl without salt, or
Bullermilk – 1 glass without salt.
Oil for cookinig – 2 tsp. only.

Aflernoon : Light tea – 1 cup with sugar 1 tsp.
Idlies 2 or dhokala 3 – 4 pieces, or Muthiya (baked), or upma, or pohe - 1 serving.

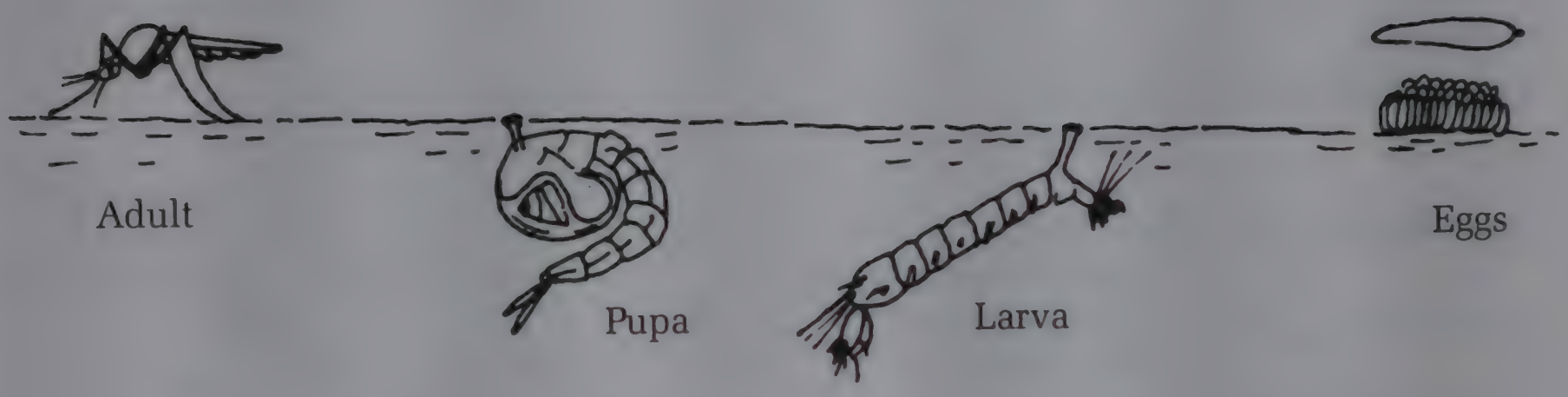
Evening : Orange – one.

Dinner : Fulka – 4 small, thin.
Khichadi – 1 medium bowl
Kadhi – 1 medium bowl
French beans vegetables – 3/4 medium bowl.

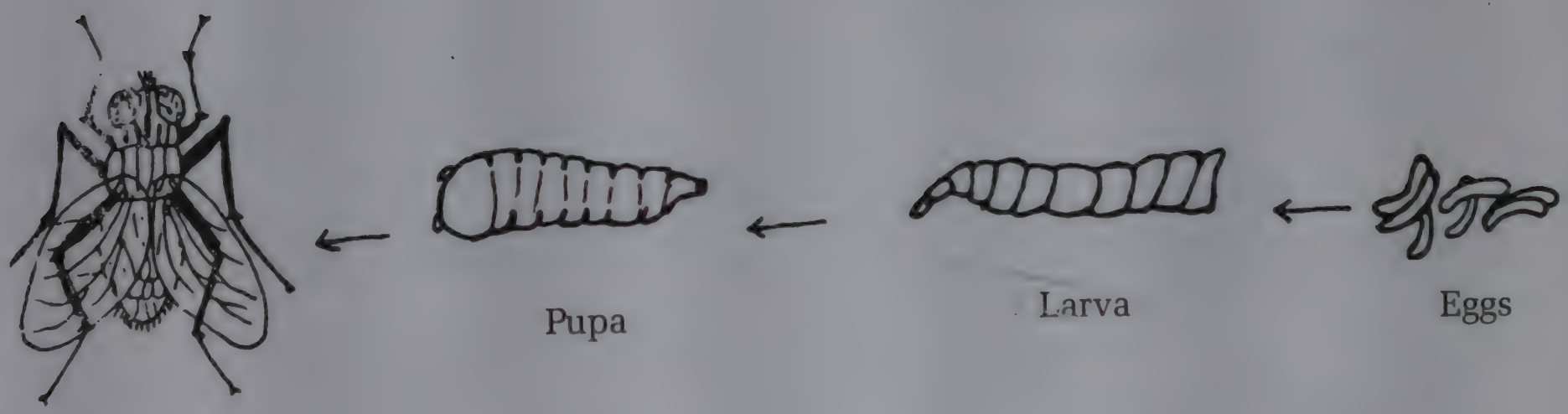
The diet provides 2,000 calories, 62 gms proteins, 45 gms fats, 330 gms Carbohydrates, 230 mg sodium and 2,500 mg potassium.



ANOPHELES



CULEX



HOUSEFLY LIFE CYCLE

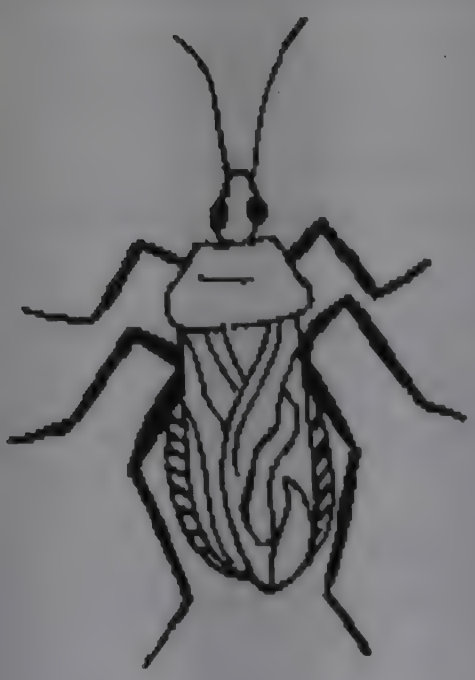
2. Insects

This is another aspect of PSM that the students never try to understand and hence find it most uninteresting. If you take into consideration the fact, that the insects are very much a part of the transmission cycle of many diseases and that their elimination will help in the control and eradication of these diseases, then you will realise the importance of knowing these insects. For the practical examination however it is enough if the student knows the diseases spread by these insects and the control measures for these insects.

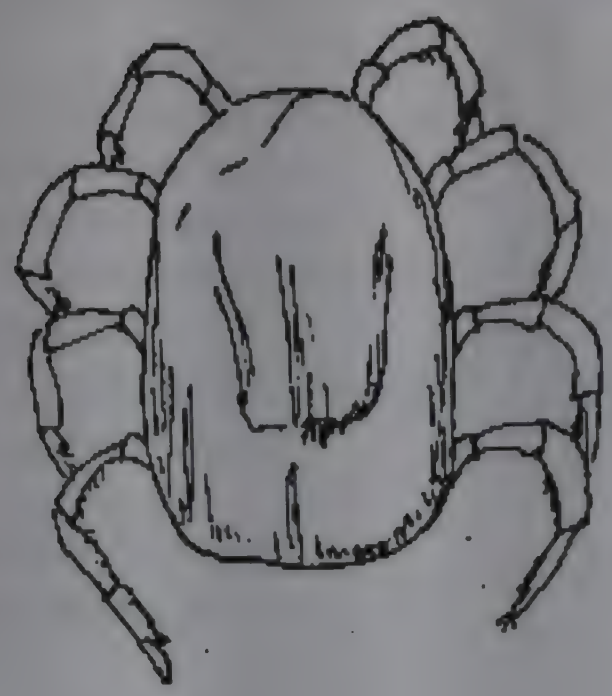
Name of the Insect	Diseases spread	Control Measures
To Mosquito:		
1. Anopheles.	a) Malaria. b) Filaria.	1. *Anti-larval measures* a. Source reduction: i) Filling, leveling and drainage of breeding places, soakage pits. ii) Rendering the water unsuitable for mosquito breeding. b. Chemical control: i) Mineral oil. ii) Paris green. iii) Synthetic Insecticides (Abate). c. Biological control: Breeding of certain varieties of fish eg. American Guppies, Gambusia affinis, Lebister reticulates, which feed voraciously on the larvae.
2. Culex.	a) Bancroftian filariasis b) Japanese Encephalitis c) Viral arthritis. d) West nile fever.	2. *Anti-Adult measures :* a. Residual spray: DDT, BHC spraying. b. Space spray: Pyrethrum, Malathion. C. Genetic control: i) Sterile male technique. ii) Cytoplasmic incompatibility. iii) Chromosomal translocation. iv) Sex distortion. v) Gene replacement.
3. Aedes.	a) Yellow fever. b) Dengue. c) Dengue hemorrhagic fever. d) Chikungunya.	3. *Protection against Mosquito bites:* a) Mosquito nets. b) Screening of rooms and buildings. c) Repellents – creams. – smokes. – odours.

Name of the Insect	Diseases spread	Control Measures
II. Houseflies:	1. Typhoid and Paratyphoid. 2. Diarrhoeas and dysentcries. 3. Cholera and gastroenteritis. 4. Amoebiasis and Giardiasis. 5. Helminthic infestations. 6. Poliomyclitis. 7. Anthrax 8. Yaws. 9. Trachoma.	1. Environmcntal control: (Eliminating brceding places) a) Collcction of Garbage, Kitchen wastes and other refuse in bins with tight lids and plastic bags. b) Proper collection, removal and disposal of refuse by incincration, composting or sani tary landfill. c) Provision of sanitary latrines. d) Stopping open air defaccation. e) Sanitary disposal of animal excreta. 2. Insecticidal control: a) Residual spray of DOT, methoxchlor, or chlordane. b) Poisoned bait. c) Cords and ribbons impregnated with insecticide. d) Space spray containing pyrethrin and DDT. e) Larvicides to be used at breeding sites. 3. Fly papers. Sticky fly paper is used. 4. Protection against flies: Screening of food articles. 5. Electronic fly catching devices.
III. Sandfly :	1. Kalaazar - Systemic and Dermal leishmaniasis 2. Sandfly fever 3. Oraya fever	They can be easily controlled, as they do not move around for long distances from their place of their breeding. 1. *Insecticides :* DOT, BHC, Malathion, Pyrethrum, spraying should be done, in the human dwellings, cattle sheds and other places. 2. *Environmential control measures :* a. Filling up cracks and crevices in walls and floors. b. Location of cattle sheds and poultry houses should be at a far distance from human habitation. c. Removal of shrubs and vegetation within 50 yards of human dwelling.

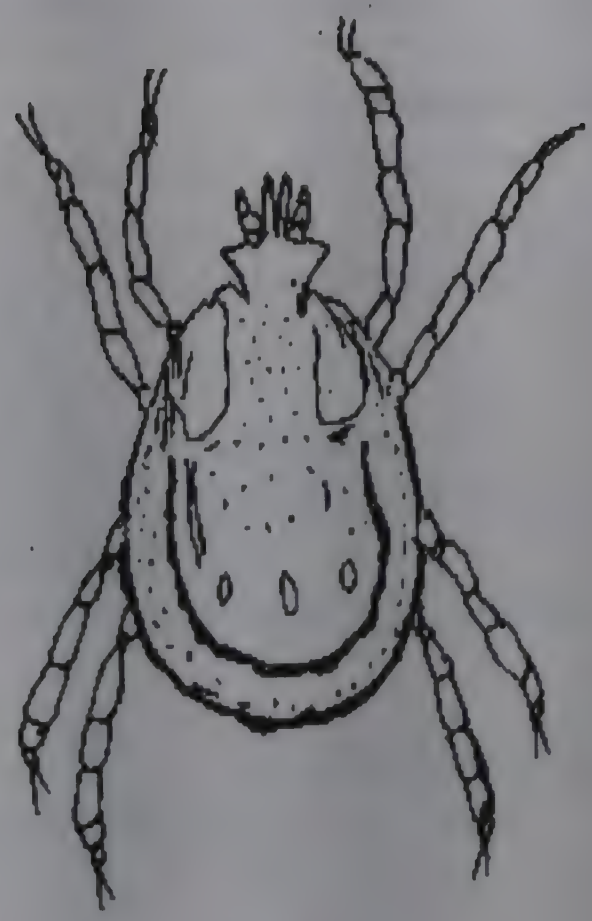
Name of the Insect	Diseases spread	Control Measures
IV. Lice:	1. Rickettsia Prowazcki. 2. Borreclia recurrentis. 3. Rickettsia quintana.	1. *Insecticidal control :* Single application of lotion containing 0.5% malathion is sufficient. 2. *Personal hygiene* : a. Daily bathing is a must and long hair should be washed and cleaned properly b. Clothing, towels and sheets should be washed in hot water and soap and ironed if possible.
V. Cyclops:	1. Dracunculosis or Guinea worm disease. 2. Diphyllobothrium latum infestation. (fish tapeworm).	1. *Physical method* : a. Straining of water through a fine cloth. b. Boiling. 2. *Chemical method :* a. Chlorine in the strength of 5 ppm b. Lime, 4 gm per gallon of water. c. Abate: I mg/litre. 3. *Biological:* a. Barbel fish and gambusia fish feed on cyclops. b. Abolition of step wells and provision of sanitary wells. c. Provision of piped water supply, Tube wells and hand pumps for drawing well water.
VI. Rat flea:	1. Plague (bubonic) 2. Endemic or murine typhus. 3. Chiggerosis. 4. Hymenolepis diminuta.	1. *Insecticidal control.* a. 10% DDT dusting of rat burrows. b. Carbaryl or diazinon. 2. *Repellent :* Diethyl toluamide (DEET) spray is an efficient flea repellant. 3. *Rodent control :* a. Sanitation measures (Rodent proof storage of eatables) b. Trapping. c. Rodenticides.



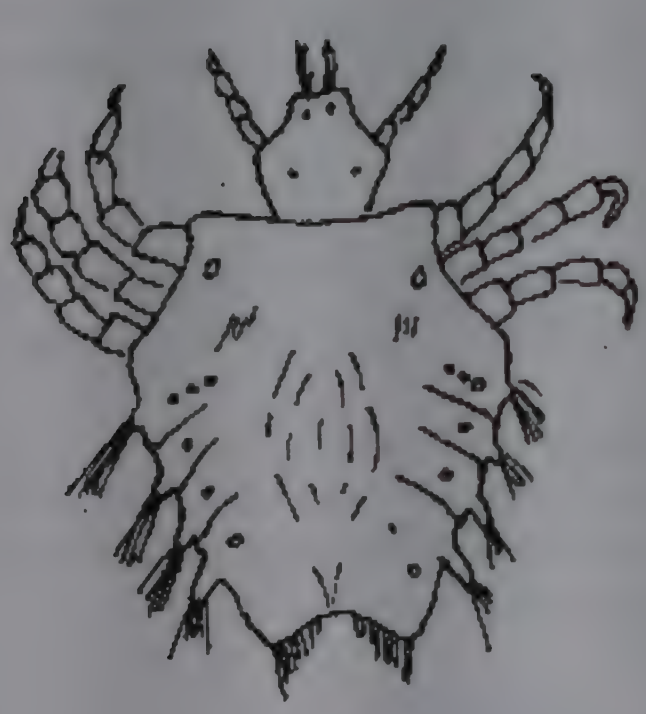
Reduvid Bug



Soft tick

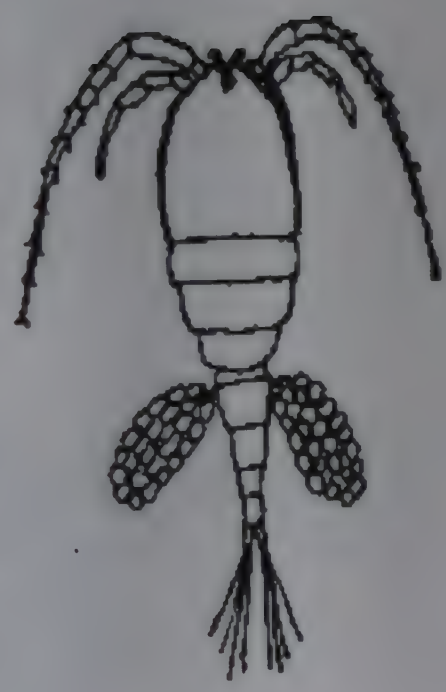


Hard tick



Phthirus pubis

Testse fly



Cyclop

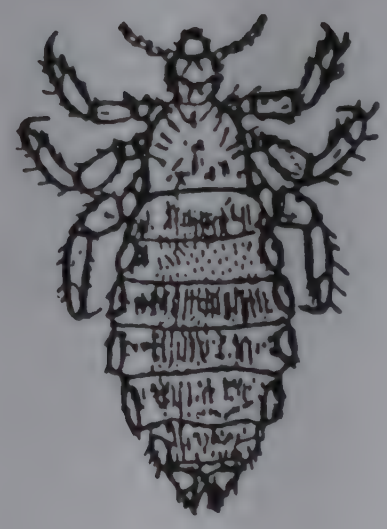
Name of the Insect	Diseases spread	Control Measures
VII. Sarcoptes scabiei or Itchmite or Acarus scabiei:	 scabiei	1. All members of the affected household should be treated simultancously. 2. Proper eleanliness and hygienc should be maintained. 3. Clean and Ironed clothes should be used. 4. Drugs: Benzyl Benzoate (25%) – Gamma benzene hexachloride (10%) – Tetmosol (5%) – Suphur ointment. (2.5 to 10%)
VIII. Trombiculid mite :	Scrub typhus	1. *Insecticidal control :* Dusting or Spraying of DDT, Chlordane, indane or Malathion on the clothing of the at-risk group. 2. *Environmental control:* Cracks and Crevices in the walls and ground should be filled up. 3. *Protection of workers:* Exposed workers should wear clothing impregnated with insect repellent.
IX. Cockroaches :	Enteric Pathogens like 1. Salmonella (enteric fever and enteritis). 2. Amocbiasis and Giardiasis. 3. Shigella dysentery.	*Chemical control :* DDT, HCH, Malathion, Pyrethrum, Diazinon, Propuxur (Baygon) can be used as a spray or bait. *Environmental contral :* Cracks and crevices in the walls, floor, furniture and fixtures, particularly in the kitchen and toilets should be filled up.
X. Reduviid Bug:	Chagas disease (Trypanosoma cruzi)	1. Residual spraying with HCH and Dieldrin. 2. Clearing of vegetation.
XI. Tse Tse Fly :	African sleeping sickness (Trypanosoma brucci)	1. Spraying with DOT, HCH and Dicldrin (Endrine). 2. Clearing of vegetation. 3. Genetic control, sterile male technique.

Name of the Insect	Diseases spread	Control Measures
XII. Hard tick:	1. Rocky Mountain Spotted fever (Tick Typhus) 2. Viral Encephalitis 3. Viral fever (Colorado tick fever) 4. Viral haemorrhagic fevers (KFD in India). 5. Tularemia. 6. Tick paralysis. 7. Human habesiosis.	1. Insecticidal control - DDT/BHC/Malathion, focal spraying in the forests. 2. Environmcntal control : - Clearing of vegetation. 3. Protection of forest workers; a) Using repellant creams and full clothing. b) Dusting of clothes with DDT/BHC.
XIII. Soft tick:	I. Q fever. 2. Relapsing fever 3. KFD.	Same as above.

Sand fly

Mite

Rat flea



Louse

3. Dis-infectants

No.	Dis-infectant	Characteristics	Mode of Action	Dosage of Chemical
1.	Bleaching powder or Chlorinated lime	a. White amorphous powder b. It has a pungent smell of chlorine	It is a chemical dis-infectant. Bactericidal and Virucidal.	5% bleaching powder is used (3 to 4 tablespoons to 1 litre of water). It is suitable for disinfeetion of faeces and urine within 1 hour.
2.	Cetrimide (cetavlon)	1. It is a yellowish solution 2. Soluble in water 3. It has soapy feel	It is strongly bactericidal against vegetative gram positive organisms but much less against gram negative organisms.	Used as a skin disinfectant.
3.	Savlon	1. It is a combination of cetavlon and hibitane (chlorhexidine)	Same as above	Plastic appliances like lippes loop may be disinfected by keeping them in it for 20 minutes.
4.	Dettol (Chloroxylenol)	It is a brownish solution.	1. It is a non-toxic antiseptic. 2. It is active against Streptococci but useless against some gram negative bacteria.	5% Dettol is suitable for disinfection of instruments, and plastic equipment It requires 15 minutes for disinfection.
5.	Halozone tablets OR Chlorine tablets	White tablets	Bactericidal and Virucidal	4mg of halozone is sufficient to disinfect about 1 litre of water in about 1/2 to 1 hour.
6.	Iodine	1. It is orange red in colour 2. It stains the skin.	1. Bactericidal and Virucidal. 2. Moderate activity against spores.	Plastic appliances like lippes loop may be sterilied by 1/2500 aqueous solution of iodine. (20ml of 2% iodine in 1 litre of distilled water.) 2.Used as a skin disinfectant (preoperatively).

No.	Dis-infertant	Characteristics	Mode of Action	Dosage of Application
7.	Phenol (carbolic acid)	1. Yellowish fluid 2. Strong pungent odour.	1. Bactericidal, causes lysis of the bacterial cell.	1.Carbolic acid and its derivatives are used as routine disinfectants of floors and other surfaces. 2.Plastic appliances can be disinfected by keeping in phenol for 20 mins.
8.	Formaldehyde (Fonnalin)	Liquid and gas with a strong irritating odour, causes burning sensation in eyes.	1. Bactericidal 2. Virucidal 3. Sporicidal	1.10% Formalin is used for sterilizing instruments. 2.Fumigation 3.Preservation of specimen and cadavers.
9.	Denatured spirit (Methyl Alcohol)	White colourless liquid with a strong odour.	1. Bactericidal 2. Fungicidal 3. Not virucidal and sporicidal	1.Used as a skin disinfectant. 2.Disinfectant for surfaces. (eg Lab. furniture)
10.	Alum	White crystalline solid or powder with a strong odour.	Collects large particles by chemical coagulation, which later settle down as a flocculate. Weak bactericidal action.	Used for removing larger organic particles before chlorination of water.

4. Insecticides

Insecticide	Consistency Odour/Colour/	Mode of action	Dosage of Application	Used against following insects	Period of effectivity
1. DDT Dichloro-diphrnyl trichlorocthan (organocillori-ne compound)	a. White amorp-hous powder b. mild, but not unplcasant smell.	It is a Contact poison (acts on the nervous system of insects It take several hours to kill.	5 percent suspension of DOT at a dosage of 100 to 200mg per square foot	1. Lice 2. Fleas 3. Ticks 4. Bugs. 5. Flies	Residual action, supposed to last as long as 18 months.
2. BHC Benzene hexachloride OR Hexachloro-cyclohexane. OR Gammexane OR Hexidol. (o-c compound)	a. White or Choc-olate coloured powder b. Musty smell. c. It is irritant to eyes, nose and skin.	It is a Contact poison.	25 to 50mg per sq. foot	Same as above	Residual action lasts for 3 months
3. Abate (Temephos) (organophos-phrous compound)	a. Brown viscous liquid	Contact poison	Not greater than 1.0 ppm	Mosquito larvae	Residual action for one month.
4. Malaltion. (o-p compound)	a. Yellow or clear brown liquid mg. b. It has an unplea-sant smell.	Contact poison	100 to 200 per sq. foot	1. Adult mosquitocs 2. Fleas 3. Ticks. 4. Bugs. 5. Flies.	Minimal residual action.
5. Pyrethrum (plant extract)	Colourless liquid or crystals.	Contact poison which kills instantly	1 oz of the spray solution per 1000c. feet of space.	1. Adult mosquitoes 2. Other Insects.	No residual effect.

Insecticide	Consistency Odour/Colour/	Mode of action	Dosage of Application	Used against following insects0	Period of effectivity
6. Paris green or Copper Aceto-arsenite	It is an emerald green erys-talline powder.	It is a stomach poison	Applied as 1 % granules to the water in which breeding occurs	1. Anopheline larvae 2. A.gambiae. 3. Other Insects.	One week.
7. Mineral Oil. such as Kerosene, Crude oil or Malariol.	Colourless or muddy liquid with a petroleum odour.	It is a contact poison.	Mosquito larvae	As long as the water surface is not disturbed	
8. Diazinon	1. It is a volatilc liquid. 2. Fumigant action	Contact poison	60 to 100 mg per sq. foot	1. Flies. 2. Mosquitoes. 3. Other insects.	
9. Fenthion	1. Brown liquid 2. Slight smell of garlic 3. It is insoluble in water.	Contact poison	20% 40% fenthion at dose of 100mg/sq. foot	1. Larvae of C. fatingans 2. Other Insects.	

5. Rodenticides

Rodenticide	Characteristics	Mode of action	Dose
1. Barium Carbonate.	White tasteless powder.	It is a single dose rodenticide but it is a weak rodenticide as compared to others. On eating the bait, rats are killed in 2 to 24 hours.	It is mixed with wheat or rice flour in the proportion of 1 part to 4 parts of flour. The mixed material is moistened with water and made into small round marbles.
2. Zinc phosphide	1. It is a greyish powde 2. It has a garlic odour.	It is a single dose rodenticide. Rats are killed in about 3 hours.	1 part of Zinc phosphide to 10 parts of wheat or rice flour. mixed with a few drops of edible oil in order to render it more attractive to the rats.
3. Warrarin	White crystalline powder, turns brownish-yellow on keeping.	It is a multiple dose (Cumulative) poison, which causes internal haemorrhage and slow death in 4 to 10 days.	Mixed with wheat or rice flour 1 : 10 parts and moistened with oil.
4. Cynogas Powder	White/brownish amorphous powder.	It gives off hydrogen cyanide gas when moistened, which is lethal to both rats and their fleas. The gas is very toxic to other animals and humans also.	It is prepared in powder form and is pumped into the rat, burrow by a special foot pump (cynogas pump) 2 ounces of poison are pumped into each burrow after moistening it and closing the exit opening.

6. Microscopic slides

Microorganisms which cause diseases of public health magnitude should be identified and certain important characteristics related to their identification, special stains and special media are expected to be known by the students. The diseases caused by these microorganisms, their signs and symptoms, their management as well as prevention and control should also be known to the students.

Important aspects related to the identification, stains and media are mentioned here. For the disease aspects the student is advised to refer back to the description or these diseases in the chapter related to case examination.

Mycobacterium Tuberculosis

Identification:

Mycobacteria means 'Fungus like bacteria'

1. It is an Acid fast bacillus (AFB)
2. They are slender rods that sometimes show branching filamentous forms resembling fungal mycelium.
3. AFB appear Pink (Magenta) against a blue background in the Ziehl Nelson stain (20% sulphuric acid).
4. They are aerobic, non-motile, non-capsulated and non-sporing.
5. They are straight or slightly curved rods occuring singly, in pairs or in small clumps.
6. Solid media for growth includes
   i) Lowenstein - Jensen media.
   ii) Loeffler's serum slope.
   iii) Dorset Egg media.

Mycobacterium Leprae

Identification:

1. They are acid-fast bacilli seen singly and in groups, intracellularly or lying free outside the cells.
2. They frequently appear as conglomerates the bacilli being bound together by a lipid like substance, the glia. These masses are known as "globi".
3. The parallel rows of bacilli in the globi present a 'cigar bundle' appearance.
4. Mycobactcrium leprae appear Pink against a blue background in the Ziehl Nelson (5% sulphuric acid) stain.

Treponema Pallidum

Identification:

1. 'Trepos' meaning to turn and 'nema' meaning thread.
2. They are slender spirochactes with fine spirals and pointed or rounded ends.
3. They are thin, delicate, spirochaete with tapering ends, and have about ten regular spirals which are sharp and regular at an interval of about 1μ. (micron).
4. They are motile, exihibiting rotation round the long axis, backward and forward. movements and flexion of the whole body.
5. It stains light rose red with Giemsa stain and can be stained by silver impregnation.

Neisseria Menningitidis

Identification:

1. They are Gram Negative, aerobic, capnophilic, non-sporing, non-motile cocci, which are both intra and extra cellular.
2. They are spherical cocci arranged in pairs, with the adjacent sides flattened.
3. Culture media are,
   a. Blood agar.
   b. Chocolate agar.
   c. Mueller-Hinton media.

Clinical features:

1. Meningococcemia:
   a. Sudden onset presentation
   b. Prodromal symptoms like cough, bodyache, headache, myalgia.
   c. Fever with chills, tachycardia, tachypnoea and shock.
   d. Petechial rash (75%)

2. Meningitis:
   a. Fever, headache and vomiting.
   b. Altered sensorium and convulsions.
   c. Neck stiffness with other meningeal signs.

Complications:

a. Addisson's crisis (Waterhouse Friedreichson syndrome.)
b. Deafness and other neurological damage.
c. Disseminated Intra-vascular Coagulation (DIC)

Treatment:

1. Antibiotics:
   a. Benzyl penicillin 10-20 lacs units Intravenous 2 hourly.
   b. Chloramphenicol 500 mg IV, 6 hourly.
   c. Third generation Cephalosporines like Cefatoxime.
2. Treatment of raised Intracranial tension with Mannitol.
3. Treatment of shock :
   a. IV fluids.
   b. Steroids.
   c. Electrolyte imbalance correction.
4. Treatment of Addisson's crisis
   a. Saline infusion.
   b. Hydrocortisone.

Corynebacterium diphtheriae

Identification:

1. They are Gram-Positive, non-acid fast, non-motile rods with irregularly stained segments and metachromatic granules and polar bodies.
2. They are non-sporing, and non-capsulated.
3. They show club-shaped swelling, (coryne meaning club), arranged in pairs or small groups and form various angles with each other so as to resembles 'V' or 'L' letters (Chinese letter arrangement).
4. Culture media are,
   i) Loeffler's serum slope
   ii) Meleod's and Hoyle's media.
   iii) Potassium Telurite blood agar

Neisseria Gonorrhoeae

Identifications :

1. They are Gram-Negative, aerobic, non-sporing, non-motile cocci.
2. They are diplococci with adjacent sides concave, the cocci are renifonn or bean shaped.
3. They are found predominantly within the polymorphs, some cells may contain as many as hundred cocci. Extracellular forms are also seen.
4. Culture media are,
   a. Blood agar.
   b. Chocolate agar.
   c. Thayer Martin medium.

Clostridium Tetani

Identification :

1. They are Gram-Positive, non-motile anaerobic, spore - forming bacilli.
2. Spores are spherical and placed terminally giving a 'drumstick appearance'.
3. The spores are wider than the bacillary bodies, giving the bacillus a swollen appearance resembling a spindle (Kloster means a spindle) hence the name clostridium.
4. Grown in anaerobic conditions only. Special medium used is Robertson's cooked meat broth.

Clinical features:

1. Inability to open the mouth (Trismus)
2. Inability to swallow.
3. Repeated fall in case of children due to stiffness of muscles.
4. Risus Sardonicus (facial muscle contraction giving the appearance of a smile)
5. Spatula test becomes positive.
   On touching the spatula to the soft palate, patient forcefully closes the mouth.

Complications :

1. Laryngeal spasm.
2. Secretions and chest infection.
3. Autonomic nervous system disturbance like cardiac arrythmia, hypotension

Treatment :

1. Anti-tetanus serum (ATS) is given intravenously, dose 5000 to 10,000 units.
2. Antibiotic: . Benzathine Penicillin is given 0.6 to 1.2 megaunit intramuscularly.
3. Muscle relaxants:
   a. Diazepam
   b. Chlorpromazine
   c. Barbiturate.
   d. Baclofen
4. Ryles tube feeding.
5. Local wound or infection site should be cleaned and antiseptic applied.

Leptospira

Identification:

1. They are elongated, motile, flexible bacteria that are twisted spirally round the long axis.
2. They possess numerous coils, set so close together that they can be distinguished only under dark ground illumination in the living state or by electron microscopy.
3. Culture media are
   a. Stuart's medium.
   b. Fletcher's medium.

Clinical features:

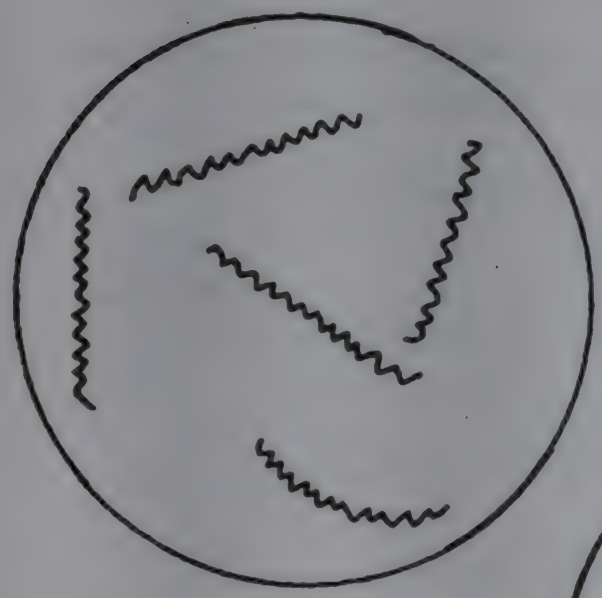
1. Leptospirosis.
   a. Primary phase: Fever, headache and joint pain.
   b. Immune or secondary phase:
      i) Signs and symptoms of meningeal irritation occurs.
      ii) Optic neuritis.
      iii) Myelitis.
      iv) Peripheral neuropathy.
2. Weil's syndrome
   a. Fever.
   b. Jaundice.
   c. Hepato-renal failure.
   d. Sub-conjunctival haemorrhages.

Treatment:

1. Benzyl penicillin 20 lacs units Intra venous, 6 hourly.
2. Fluid and electrolyte imbalance to be corrected.
3. Blood transfusion if required.
4. Renal dialysis if required.

Note : Leptospirosis is transmitted by rats and is an occupational huzard of sewage drainage workers.

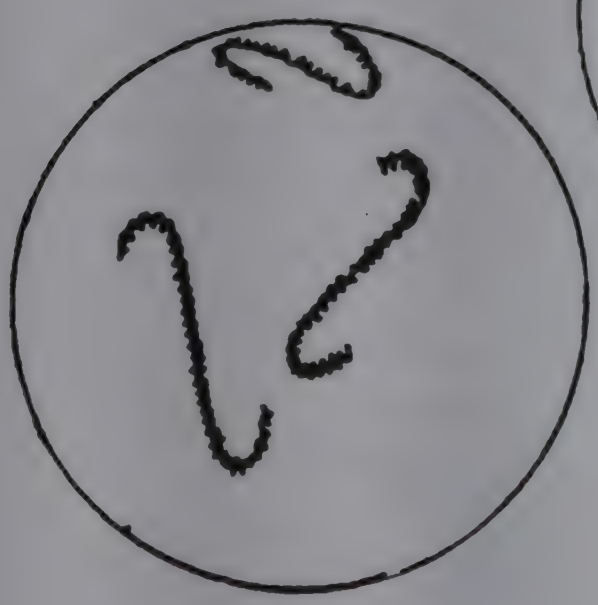
T. Pallidum



Leptospira

C. diphtheria

M. Lepre



Mycobacterium Tuberculosis

7. Contraceptives

Coatraceptive methods are available for the control of population. Unless one understands the exact technique of use, advantages and disadvantages it is not possible to motivate eligible couples to use them. Every doctor is expected to know the use of contraceptives thoroughly.

In PSM practicals one contracrptive method is invariably kept, the student is usually expected to identify the contraceptive and then write a few lines on its advantages, disadvantages and failure rate.

Definition:

These are the methods/measures, temporary or permanent, designed to prevent pregnancy.

An Ideal Contraceptive method should have following characteristics.

a. Widely acceptable.
b. Inexpensive
c. Simple to use.
d. Safe to use.
e. Highly effective
f. Requires minimal motivation, maintenance and supervision.
g. Doesn't interfere with pleasure of coital act.

Contraceptive failure rate

$$= \frac{\text{No. of accidental pregnancies x 1200}}{\text{No. of Patients observed x months of use.}}$$

Example: If 100 couples have used a method for a period of 1 year and has resulted in 20 accidental pregnancies then the pregnancy rate (contraceptive failure rate) would be

$$\frac{20 \times 1200}{100 \times 12} = 20/\text{HWY}$$

∴ Pregnancy rate (contraceptive failure rate) = 20 per Hundred Women Year of exposure, (HWY)

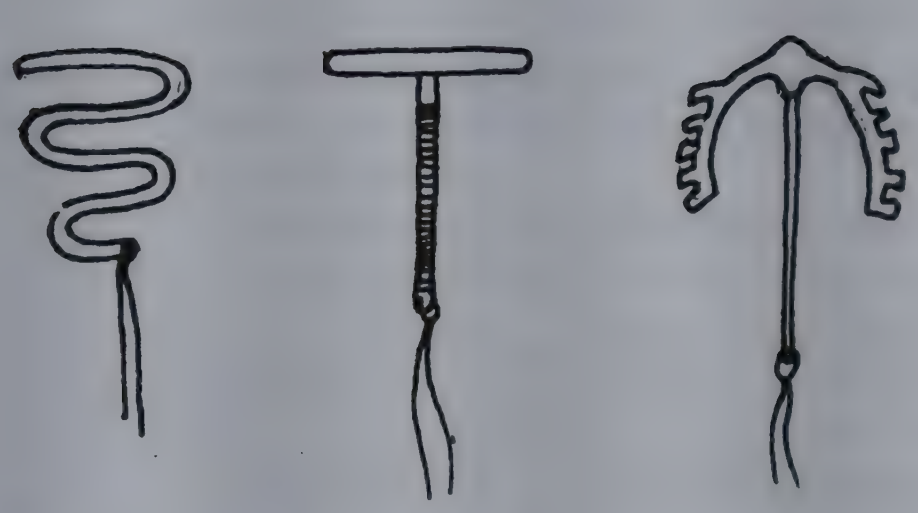
(Do not forget to write the rate in terms of HWY, or you will lose valuable marks).

Contraceptive	Advantages	Dis-advantages	Failure rate or Pregnancy Rate
1. Condom	1. Cheap and safe 2. Easy to carry 3. Simple to use, hence does not require medical supervision. 4. Light, compact and disposable. 5. Protection against venereal diseases (part of AIDS prevention measures) 6. No side effect. 7. Useful where coital act is infrequent and irregular.	1. It interferes with sexual pleasure. 2. It may slip off or tear during coitus. 3. It is for single use only (i.e. not reusable)	14/HWY
2. Diaphragm, Cervical cap, Vault Cap (Dumas)	1. Can be used for longer duration (reusable) 2. Total absence of risks and medical contra-indications.	1. Initially Doctor or Para-medical person is needed for insertion / demonstration. 2. Some training is required for proper insertion. 3. Privacy is required for insertion, washing and storing the diaphragm.	12/ HWY

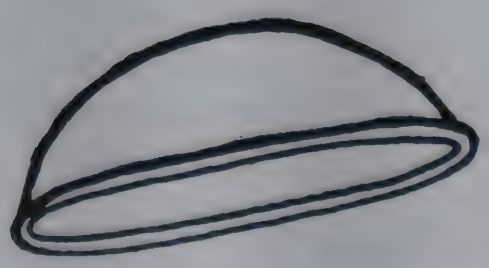
Contraceptive	Advantages	Dis-advantages	Failure rate or Pregnancy Rate
		4. It can cause Toxic Shock Syndrome. 5. Additional protection by a chemical spermicidal agent is required.	
3. Vaginal sponge (tampon parsnip)	1. Easy to insert. 2. Disposable.	1. Privacy is required for insertion. 2. More expensive. 3. It can cause Toxic shock Syndrome.	10/HWY
4. Spermicidals : a. Foam tablets. b. Cream. c. Pessary.	1. Easy to use	1. High failure rate. 2. It may cause mild burning or irritation. 3. It is messy. 4. May require condom or diaphragm in addition.	5/ HWY

Name	Advantage	Dis-advantage	Side effects	failure rate
5. OC pills (MalaD)	1. Highly effective 2. Good menstrual cycle control. 3. Reversible immediately 4. Well tolerated. 5. Also useful in associated Conditions like dysmen-orrhoea and Pre-menstrual tension.	1. Requires iriitial check-up and peri-odic examination. 2. Systemic side-effects may be harmful 3. Requires motivation. 4. Limitation in its use. (maximum contin-uous use for 3 to 5 years)	1. Nausea, vomiting, headache. 2. Mastalgia. 3. Weight gain. 4. Break through bleeding. 5. Depression. 6. Hypertension. 7. Thrombo-embolic phenomena. 8. Hepatitis. 9. Endometrial and ovarian neoplasm 10. Diabetes.	0.5 to 1/HWY.
6. Intra-uterine Contraceptie Devices. a. Cu-T b. Lippe's Loop c. Multiload	1. Inexpensive 2. Simple technique of insertion. 3. Can be used for 3 years. 4. No systemic side effects 5. Reversibility to fertility is immediate, soon after removal. 6. Better tolerated by nullipara.	1. Increased incidence of ectopic pregnancy. 2. Limitation in its use.	1. Cramp like pain. 2. Syncopal attack. 3. Abnormal mens-trual bleeding. 4. Pelvic inflam-matory disease. 5. Spontaneous expulsion.	1.5 to 3 / HWY.

7. Depot-medroxy Progesterone actate (DMPA)	1. Excellant compliance 2. Safe, effective and acceptable contraceptives 3. Minimum or no motivation is required 4. It does not affect lactation. 5. Reversible.	1. It should not be used in breast cancer and all genital malignancies 2. It should be used cautiously in abnormal uterine bleeding	1. Weight gain 2. Irregular bleeding 3. Prolonged infertility after its use.	0.5 to 2/ HWY.
8. NET-EN Norethist-crone enantate	Same as above	Same as above	Same as above	1 to 4/ HWY.
9. Norplant	1. Highly Effective 2. Safe 3. Reversible on removal of capsules	1. Surgical procedure is neccessary to insert	1. Irregularity of bleeding. 2. weight gain	0.7 HWY



Lippe's Loop Copper - T Multiload



Diaphragm

8. Vaccines

Vaccine	Composition	Dosage/Schedule	Mode of adminstration	Complications/Adverse	Contra-indications	Storage
1. Polio Vaccine Live, oral (Sabin) Trivalent	It is a mixture of three polioviruses, propagated in primary monkey kidney tissue cultures. Each dose contains Type 1: Poliovirus 300000 TCID 50 Type 2 : Poliovirus 100000 TCID 50 Type 3: Poliovirus 30000 TCID 50	Primary Immunisation: Three doses at an interval of 4 to 6 weeks starting from 6 weeks of age. Booster Immunisation: It should be given between 18-24 months. of age. Recently zero dose at birth has been introduced.	Orally	1. Vaccine associated Paralytic Polio (one /3 million vaccination)	1. Persistent vomiting. 2. Diarrhoea. 3. Upper respiratory tract infections which may interfere with the desired response. 4. Fever. 5. Leukemia. 6. Malignancy.	1. Vaccine vial can be stored at 2. 2 to 8°C far 120 days from the date of issue, or if stored at – 20°C or below then for 2 years. 2. Should not be repeatedly frozen and thawed.
2. Measles vaccine, live, lyophilised	0.5 ml contains not less than 1000 TCID50 (Tissue culture infective dose) of measles virus.	Single dose to Children at 9 months of age.	Subcutaneous	1. Minimal rise in temprature in (5-6%) 2. Mild skin rash in 1 to 2%. 3. Mild gastric disorders.	1. Febrile state. 2. Acute infectious disease. 3. Severe disease of the hemato-poietic system. 4. Severe renal impairement. 5. Decompensated heart disease. 6. Reduced immunity. 7. Within six months following exchange transfusion. 8. Pregnancy. 9. Patient on corticosteriod.	It should be stored in a dark place below 8°C.

Vaccine	Composition	Dosage/Schedule	Mode of adminstration	Complications/Adverse	Contra-indications	Storage
3. Triple Antigen Injection (Adsorbed Diptheria, Tetanus, and Pertussis vaccine)	Each dose contain Diptheria Toxoid 25Lf Tetanus Toxoid 5 Lf. Pertussis vaccine 4 international units Aluminium Phosphate 1.5 mg Thiomersal 0.01% as preservative.	Primary: Three injections of 0.5 ml at interval of 4 to 6 weeks from 6 wks of age. Booster injection of 0.5 ml should be given at 18-24 months of age.	Intramuscular	1. Pain, erythema tenderness and induration at the injection site. 2. Mild transient fever and irritability 3. Nodule at the site of injection 4. Sudden infant death syndrome (SIDS), is an extremely rare complication.	1. It should not be administered to infants or children with fever or other evidence of acute illness. 2. It should not be given during an outbreak of Poliomyelitis. 3. It should not be given to children over six years of age or adults because of the danger of reaction to diptheria toxoid or to pertussis vaccine.	1. It should be stored between 2°C to 8°C 2. It should not be frozen.
4. Dual Antigen Adsorbed Diptheria and Tetanus vaccine.	Diphtheria Toxoid 25Lf. Tetanus Toxoid 5Lf. Aluminum Phosphate 1.5mg Thiomersal 0.01 % as preservative.	1. Given as second booster after 36 months of age. 2. Also used for primary immunisation in children sensitive to pertusis components	Intramuscular	Same as above.	Same as above.	Same as above.
5. Hepatitis B	20 mgm of antigen protein. 0.5 mg Aluminium hydroxide. Thiomersal as preservative.	Immunocompetant Adult = 20 μgm (1ml) Children = 10 μgm (0.5 ml) Immunosuppressed adult = 40 μgm (2ml) 1. Primary : It should be given on 0, 1 and 6 mths. 2. Boosters every 3-5 years.	Intramuscular in the detloid region	1. Transient soreness erythema and induration at the site of injection. 2. Low grade fever malaise, fatigue arthralgia, myalgia, headache, nausea and skin rash.	Severe febrile infections.	It should be shipped under refrigeration and stored at 2°C to 8.C.

Vaccine	Composition	Dosage/Schedule	Mode of adminstration	Complications/Adverse	Contra-indications	Storage
6. Cholera vaccine (Killed vaccine)	V. cholera Inaba = 6000 million Ogawa=6000 organism per mI.	Adult 0.5 ml Male first dose then 1 ml 4 to 6 weeks later. Female: Half the adult dose. Children: One fifth of the adult dose. (Poor immunity, lasts for about 6 months)	Sub - cutaneous	1. Swelling at the site of injection. 2. Minimal rise of temperature. 3. Weakness, myalgia lethargy.	None	Storage between 2°C to 10°C and not to be frozen.
7. Typhoid	Salmonella typhi-1000 million bacteria per ml having the full complement of O, H and Vi antigens.	Adult Male: Primary inoculation 1st dose 0.5 ml 2nd dose 1 ml (4-6 wks.later) Re -inoculation (every year) 0.5 ml Female: Half the adult dose. Children: One fifth the adult dose. (Immunity lasts for 8-12 monhts)	Sub - cutaneous	1. Stinging or aching sensation at the site of injection. 2. Redness, induration and tenderness at the site of injection. 3. Rise of temprature	None	Between 2°C to 8°C not to be frozen.
8. Rabies Vaccines (Attenuated live vaccine) (HDCS)	Rabies "fixed" virus in human diploid fibroblast cell.	Total 6 injections. It should be given on 0, 3, 7, 14, 30, and 90 days.	Intra - muscular in the deltoid muscle.	1. Mild allergic skin reaction, pain at site of injection. 2. Mild fever. 3. Head-ache, lethargy.	None	Between 2°C to 8°C not to be frozen.
9. Rabipur vaccine	Purified chicked embryo cell Rab vaccine contains Inactivated rabies virus, It is inactivated with B.Propiolactone, purified by density gradient centrifugation and concentrated.	Post exposure: 0,3, 7,14,30 and 90 days.	Intra - muscular in the deltiod nuscle.	1. Pain, erythema and swelling at the site of injection (in 5% cases) 2. Allergic skin reaction	Allergy to chicken protein.	It should be stored at 2°C to 8°C.

Vaccine	Composition	Dosage/Schedule	Mode of adminstration	Complications/Adverse	Contra-indications	Storage
10. Rabies vaccine BPL inactivated. (Semple vaccine) (Sheep brain vaccine)	Sterile 5% infected sheep brain suspension inactivated with BPL in buffer solution with 0.25% phenol on thiomersal as a preservative.	The dosage depends on the classification of the wounds and the age of the bitten person. Class. I: 2 ml daily for 7 days. Class. I I : Adult = 5 ml daily for 10 days. Children =2ml daily for 10 days. Class. III : Adult = 5 ml daily for 10 days. Children = 2 ml daily for 10 days	Sub - cutaneous in the abdomen	1. Erythema, Oedema, pruritus at the site of injection. 2. Post vaccination demylination neuroparalysis (rare).	None	It should be stored in a refrigerator at 2°c to 8° c.
11. BCG (Lyophilised, attenuated, live vaccine)	Attenuated strain of mycobacterium bovis	Single dose, Children under 1 year = 0.05 ml. Above than 1 year = 0.1 ml.	Intrademlal	1. Local: a. Koch's phenomen (acclerated local reaction) b. Abscess formation c. Keloid place. d. Tuberculids 2. Focal: Abscess formation in the regional lymph node. 3. Generalised: - Fever - Erythema nodosu - Generalised Tuberculosis.	1. Cell mediated immune deficiency	1. Stored and transported between 2°C to 8°C. 2. Vaccine must be kept in a dark

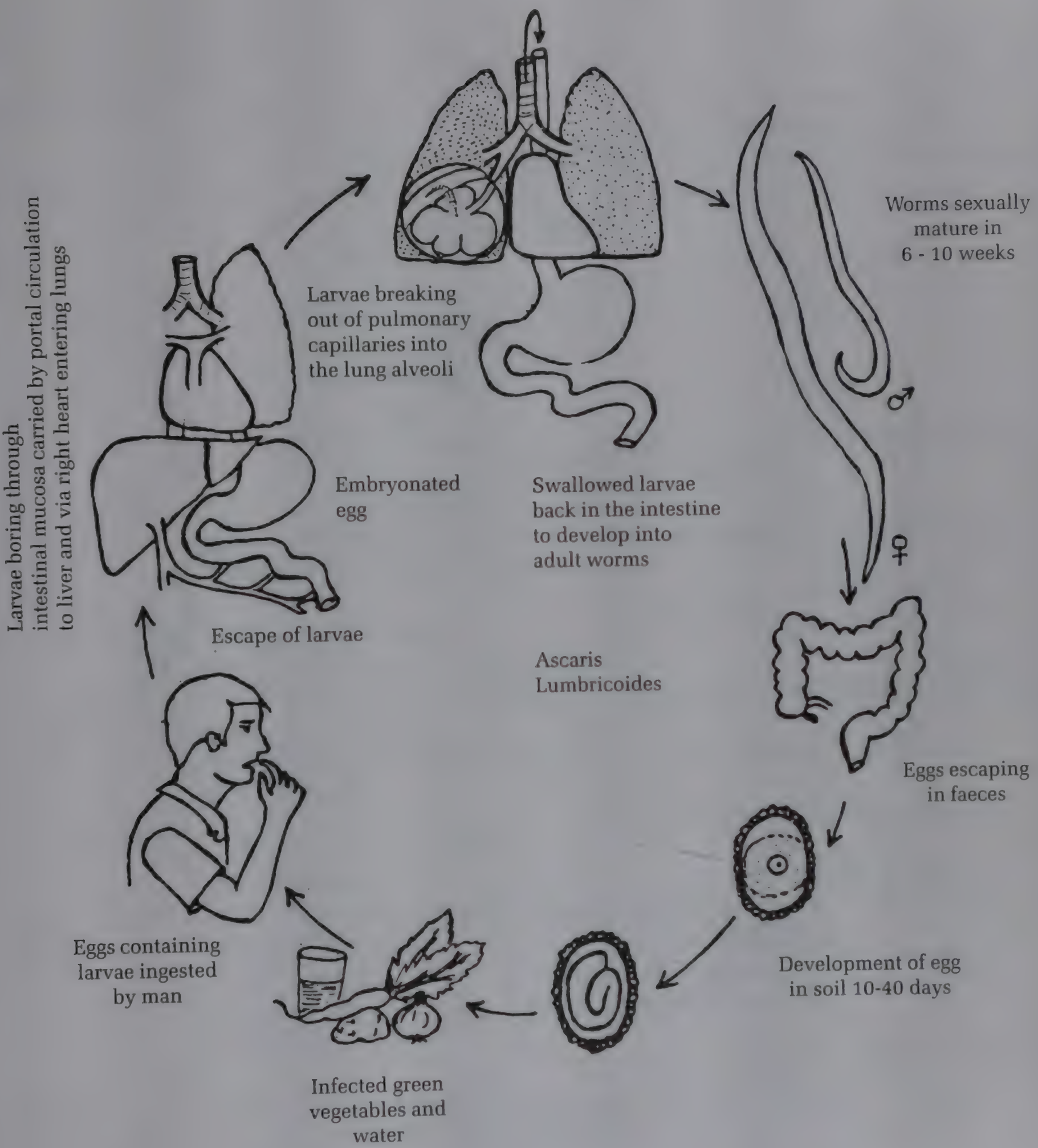
9. Helminthology

Worm infestations are responsible for a considerable amount of morbidity in our country. Intestinal helminths take away valuable nutrients and predispose to various types of malnutrition. Full size specimen of helminths are kept as part of spots in the PSM practicals. The student is expected to identify the helminth and answer a few relevant questions or even draw a sketch of its life cycle. All important practical aspects of important helminths are mentioned here in an easy to memorise form.

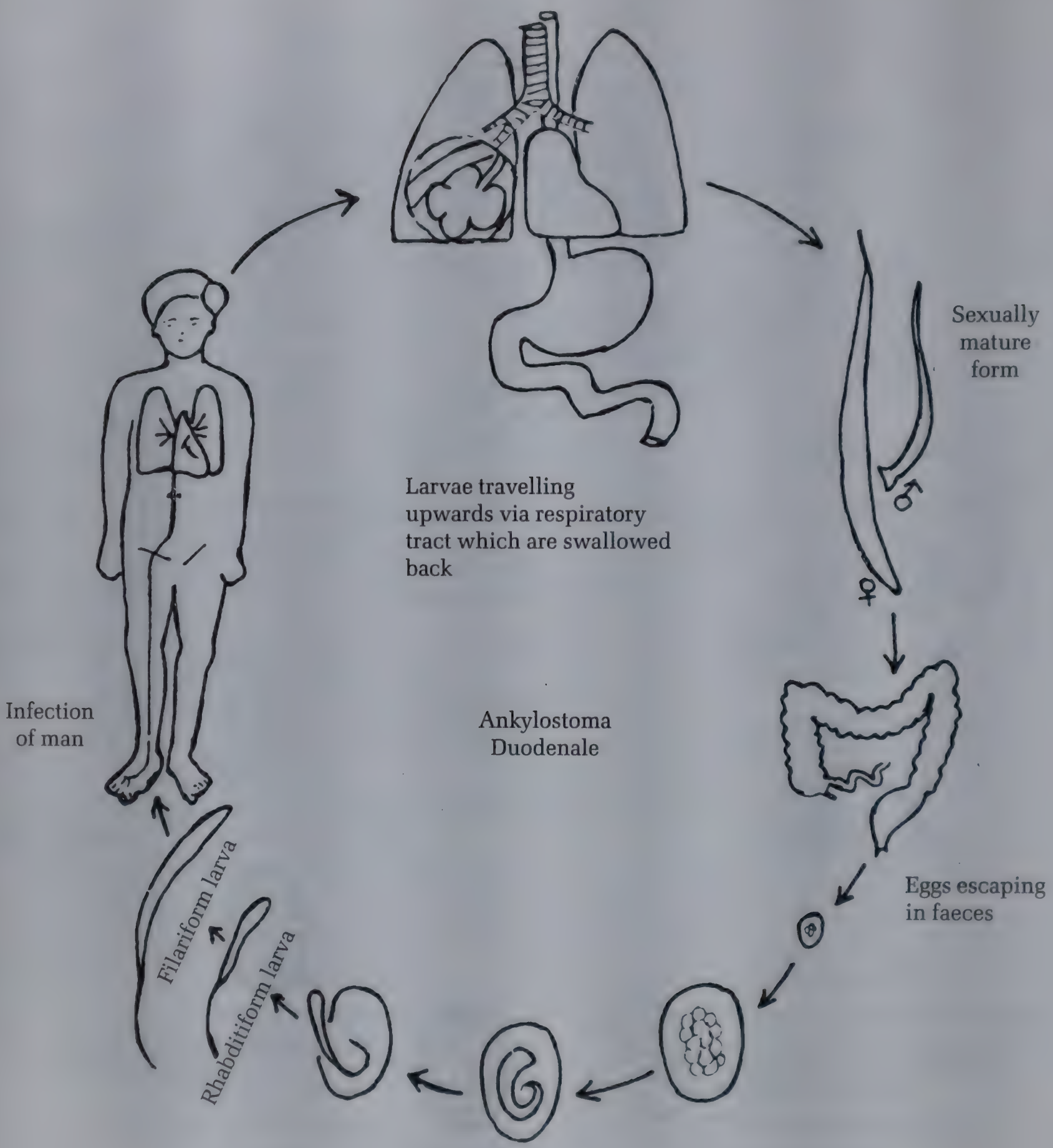
	Round Worm	**Hook Worm**
Biological Name	Ascaris lumbricoides	Ankylostoma duodenale
Location in human being	Small intestine	Small intestine.
Mode of entry	Oral: Ingestion of contaminated vegetables	Skin penetration by larvae
Life cycle	Fertilized eggs in stool ⇓ Embryonation of eggs in soil ⇓ Ingestion of contaminated vegetables ⇓ Digestion of eggs shells in duodenum ⇓ Liberation of rhahditiform larvae ⇓ Penetration of larvae in intestine ⇓ Circulation ⇓ Lungs ⇓ Break through pulmonary capillaries into alveoli. ⇓ Maturation of larvae in intestine. ⇓	Eggs with 4 blastomeres in stool. ⇓ Eggs hatch in soil. ⇓ Rhabditiform larvae. ⇓ Filariform larvae (infective to men) ⇓ Skin penetration on walking bare foot. ⇓ Venous circulation ⇓ Right heart ⇓ Lungs ⇓ Break through pulmonary capillaries. ⇓ Climb up trachea and descend into the oesoplagus ⇓

	Round Worm	Hook Worm
	Development of adult worm in intestine ⇓ Fertilization ⇓ Eggs in faeces	Intestines ⇓ Adult worms ⇓ Eggs in stool.
Manifestations	1. Passage or worm in stool or vomitus. 2. Vomiting and diarrhoea. 3. Abdominal distention. 4. Flatulence. 5. Cough. 6. Malnutrition. 7. Skin rashes. 8. Encephalopathy.	1. Abdominal pain. 2. Diarrhoea. 3. Anaemia. 4. Anorexia. 5. Dyspnoea. 6. Swelling of feet.
Diagnosis	1. Stool examination for ova. 2. Barium meal.	Stool examination for ova and for larvae.
Prevention	1. Hands should be washed before eating or handling food. 2. Vegetables should be washed thoroughly. 3. Untreated sewage should not be used as fertilizer. 4. Proper disposal of sewage.	1. Avoid walking barefoot. 2. Proper disposal of faeces
Treatment	1. Piperazine citrate. 2. Hexyl resorcinol. 3. Thiabendazole. 4. Mebendazole. 5. Albendazole. 6. Levamisole and Tetramisole. 7. Pyrantel pamoate.	I. Treatment of anaemia. II. Antihelminthic drug therapy: 1. Bephenium hydroxynaphthoate. 2. Hexyl resorcinol 3. Tetrachlorethylene. 4. Mebendazole. 5. Albendazole. 6. Thiabendazole. 7. Levamisole and Tetramisole. 8. Pyrantel pamoate.

Ascaris Lumbricoides



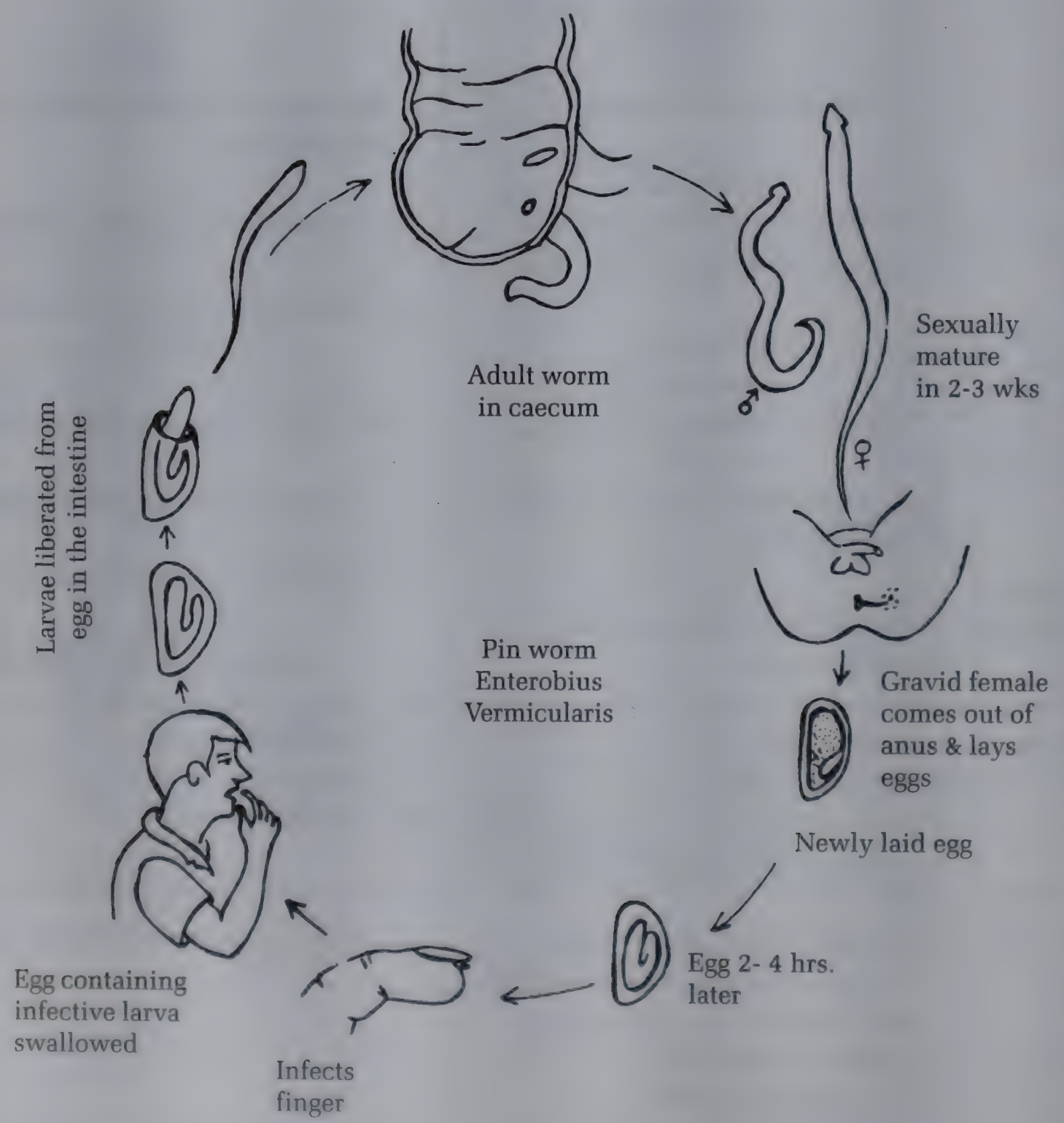
Ankylostoma Duodenale



	Pin Worm	Thread Worm
Biological name	Enterobius vermicularis	Stercuralis Strongyloides
Location in human body	Caecum and appendix, rarely vagina and fallopian tube	Small intestine
Mode of entry	Oral ingestion, from contaminated hands.	Through skin, penetration of foot by larvae while walking barefoot.
Life cycle	Embryonated eggs in finger nails ⇓ Ingested ⇓ Larvae hatch in duodenum ⇓ Sexual malurity ⇓ Fertilization in intestine ⇓ Nocturnal migration of gravid female to anal region to lay eggs ⇓ This causes perianal itching at night Eggs get embeded in the nail beds while scratching ⇓ Auto infection through contaminatcd hands.	Penetration of skin by larvae ⇓ Venous circulation. ⇓ Right heart ⇓ Lungs. ⇓ Migration to bronchi, trachea, larynx and epiglottis. ⇓ Swallowed back and enter into the intestine ⇓ Development of males and females in the intestine ⇓ Pentration of females through the mucosa. ⇓ Deposition of eggs (asexual) ⇓ Hatching of rhabditiform larvae. ⇓ Filariform larvae in faeces.
Manifestations	1. Irritabilily. 2. Perianal itching. 3. Loss of appetite. 4. Symptoms of appendicitis.	1. Abdominal pain 2. Diarrhoea. 3. Malabsorption.
Diagnosis	1. A transparent adhesive tape is applied in anal area at night and examined in the morning under microscope. 2. Tiny worms in stool	1. Stool examination for larvae.

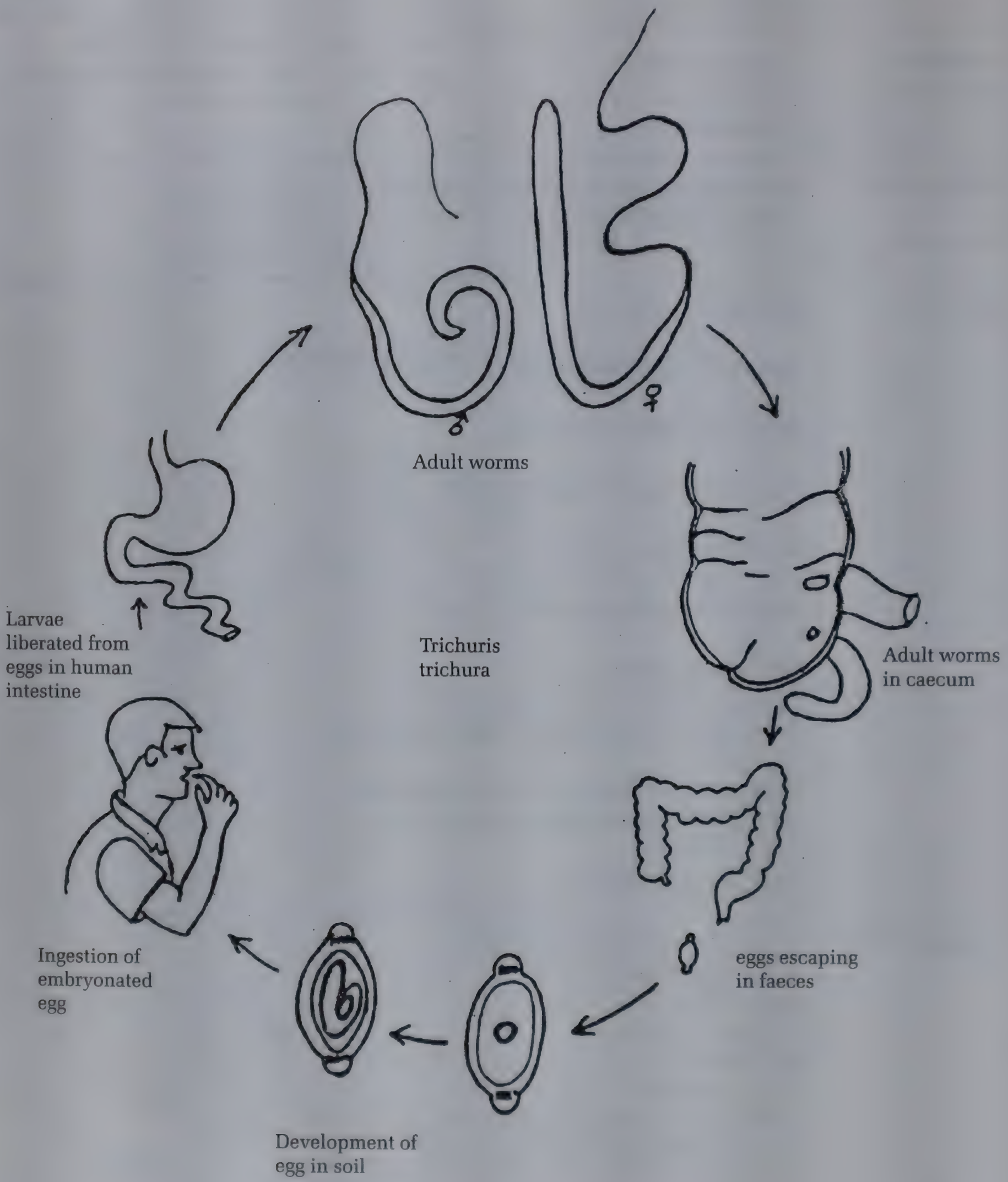
	Pin Worm	Thread Worm
Prevention	1. Personal hygiene 2. Nails to be cut and trimmed 3. Washing of nails and peri anal area with soap and water.	1. Avoid walking barefoot 2. Proper disposal of faeces.
Treatment	1. Piperazine citrate. 2. Thiabendazole. 3. Mebendazole. 4. Albendazole. 5. Pyrantel pamoate.	1. Levamisole and tetramisole. 2. Thiabendazole. 3. Mebendazole. 4. Albendazole.

Enterobius vermicularis



	Whipworm	
Biological name	Trichuris trichura	**Prevention :** 1. Personal hygiene. 2. Vegetables should be washed well 3. Efficient sewage disposal 4. Untreated sewage should not be used as fertilizer.
Location in human body	1. Large intestine 2. Caecum 3. Appendix	
Mode of entry	Oral: Ingestion of eggs through unclean vegetable, salads	**Treatment :** 1. Thiabendazole 2. Mebendazole 3. Albendazole
Life Cycle	Eggs passed in the stool ⇓ Eggs mix up with vegetables. ⇓ Ingestion of embryonated eggs. ⇓ Digestion of eggs by digestive juices ⇓ Liberation of larvae ⇓ Attachment in small intestine ⇓ Migration to caecum ⇓ Development of adult worms, male and female. ⇓ They mate in the intestine and produce eggs 5000- 7000 eggs are produced by one worm ⇓ Eggs passed in the stool.	
Manifestations	1. Asymptomatic, if light infection. 2. Vague abdominal pain. 3. Mild diarrhoea. 4. Blood in stool. 5. Tenismus. 6. Losd of weight. 7. Failure to grow, if heavy infection 8. Rectal prolapse. 9. Volvulus.	
Diagnosis	Unsegmented bile stained ova in stool which float in saturated salt solution.	

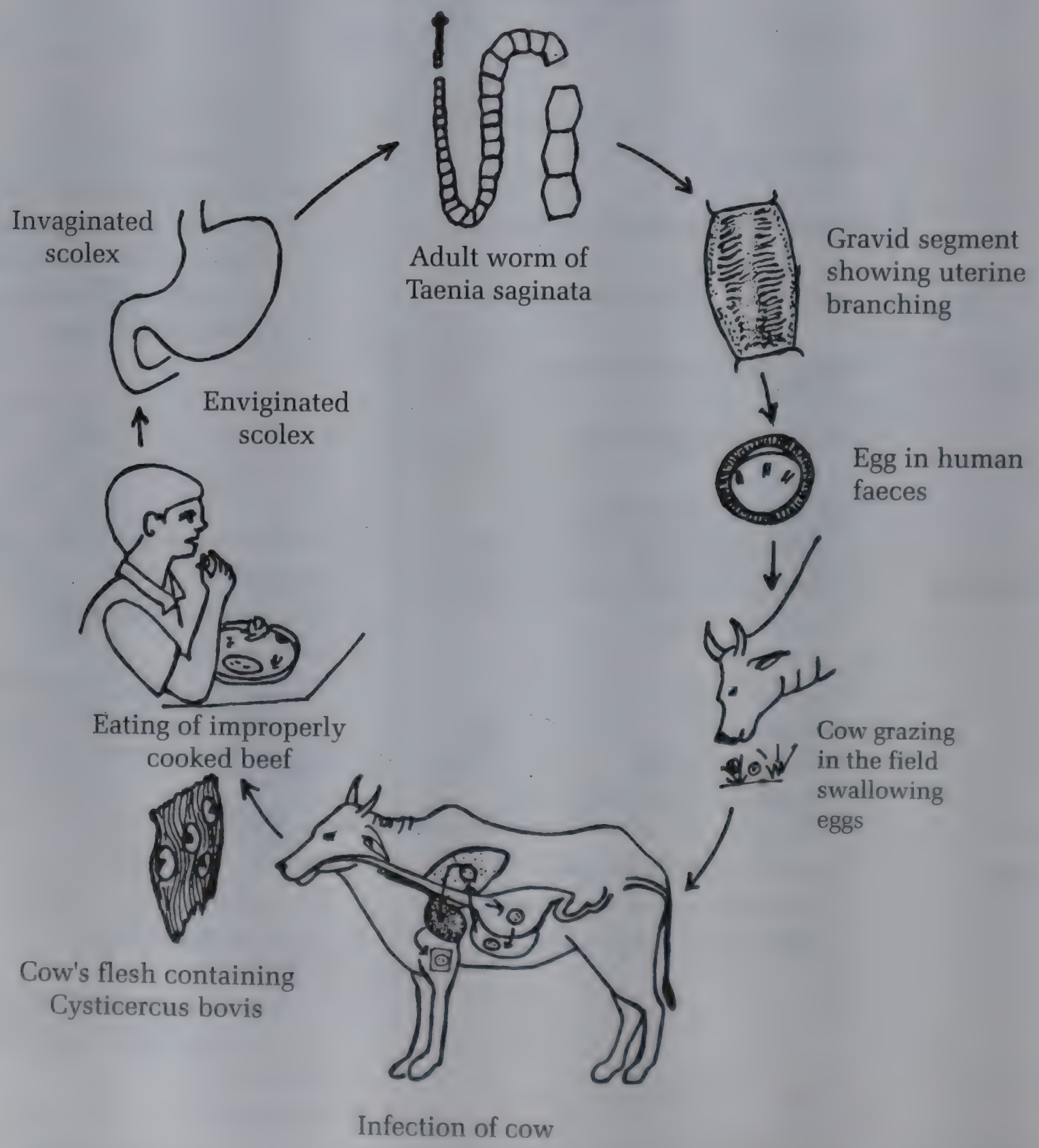
Whip worm (Trichuris trichura)



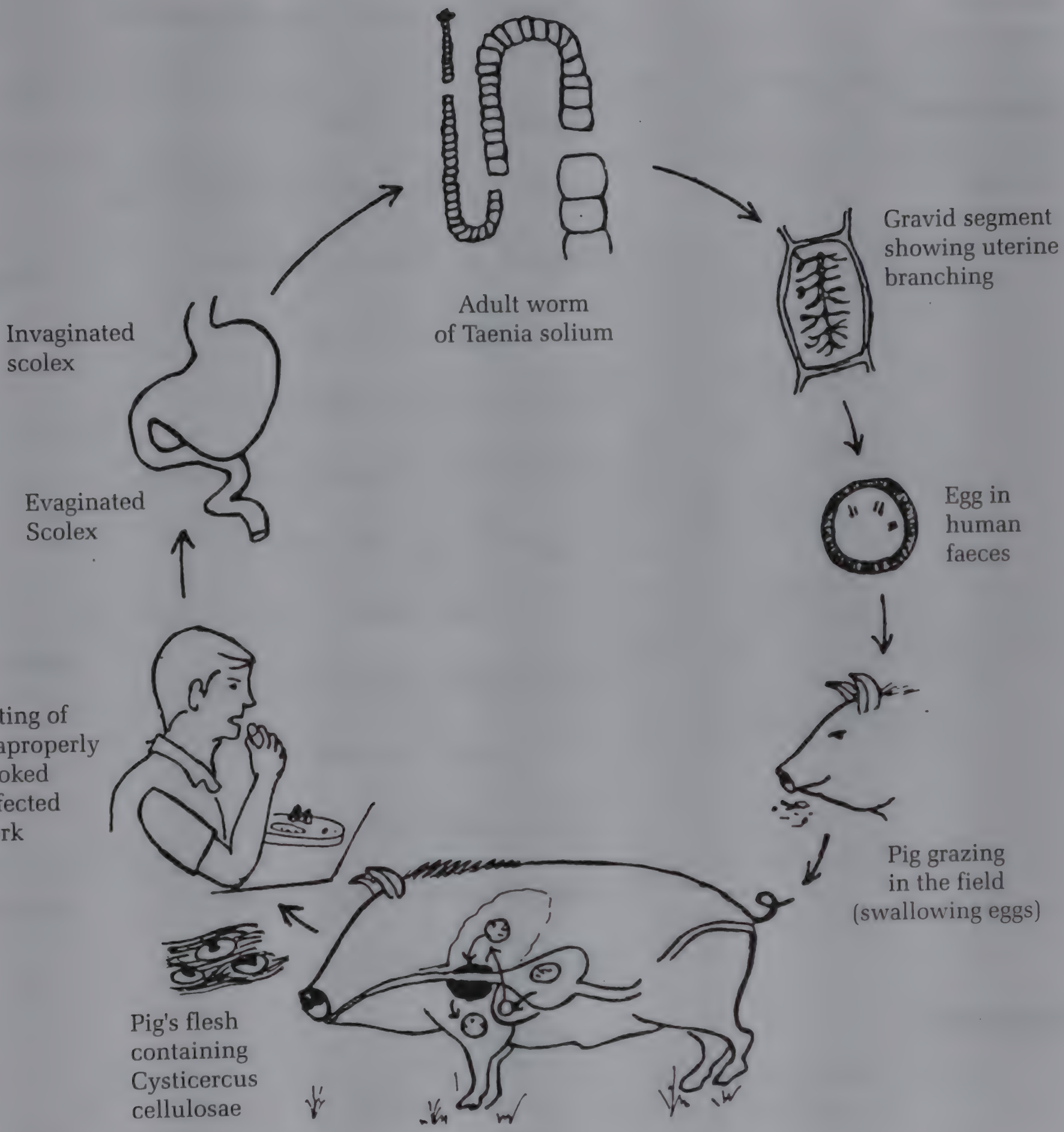
Tape worm		
Biological Name	Taenia saginata (Beef tape worm)	Taenia solium (Pork Tapeworm)
Loration in hunlan body	In small intestine, with the suckers on the scolex, they get embeded into the wall of the intestines.	Same
Mode of Entry	Eating uncooked Beef.	Eating uncooked Pork.
Life cycle	Ova in infected human faeces. ⇓ Ingestion by cattle ⇓ Liberation of oncosphere ⇓ Reaching muscles ⇓ Cysticercus bovis development within muscle ⇓ Ingestion of infected flesh by man. ⇓ Cysticercus bovis gets digested ⇓ Scolex attaches to intestines ⇓ Adult worm is produced. ⇓ Eggs are passed in stool.	I. Same (substitute pig instead of cattle and cysticercus cellulosae instead of Bovis) II. Cysticercosis: Here man serves as a larval host of T. solium instead of pig. Infection is acquired by one of these 4 mechanisms; 1. Drinking contaminated water. 2. Eating uncooked vegetables infected with eggs. 3. Autoinfection in man. Adult worm gets inside by. a. Unclean and unhygenic habits b. Reversal of peristalsis where gravid segments are thrown into the stomach, equivalent to swallowing thousands of eggs.
Manifestations	1. Passage of long ribbon shaped worm in stool. 2. Debility. 3. Loss of weight. 4. Anaemia. 5. Intestinal colics. 6. Intestinal obstruction.	1. Headache 2. Bout of unconsciousness 3. Epileptic fits. 4. Opthalmic: Iridocyclitis, retinitis, Choroiditis, palpebral conjunctivitis, cysts in ocular muscles.
Diagnosis	1. Passage and finding of extremely active egg filled segments moving in faeces.	1. Stool examination. 2. CT scan or head. 3. Immunological tests. i. CFT ii. Skin testing using fluid from C. cellulose or C. bovis antigen. 4. Biopsy. 5. Eosinophilia.

Tape worm		
Prevention	1. Carefully examine beef for cysts 2. Utilizing well cooked meat.	1. Personal hygiene. 2. Quality checking of pork. 3. Use only well cooked pork.
Treatment	1. Mepacrine 1 gm through ryles tube. 2. Niclosamide 2 tablets stat followed by 2 tablets after 10 minutes. Followcd by saline purge. 3. Albendazole 4. Bithionol.	Niclosamide: Total 2gms with saline purge. Praziquantel 50 mg/kg daily x 15 days. Albendazole 15 mg/kg daily x 30 days.

Taenia saginata



Taenia solium



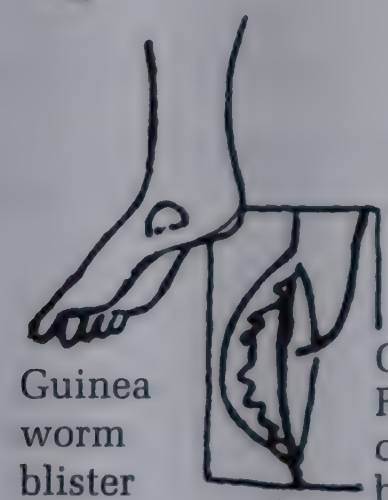
Infection of pig

	Guinea worm, Serpent worm, Dragon worm, Dracunculus medinensis.
Biological name	Dracunculus medinensis.
Location in human body	Subcutaneous tissues of legs, arms and back.
Mode of entry	Ingestion of infected water
Life cycle	Ingestion of water contaminated with cyclops. ⇓ Digestion of cyclops in the stomach ⇓ Liberation of larvae ⇓ Larval migration through intestine ⇓ Entry into retroperitoneal connective tissue ⇓ Matures into an adult worm ⇓ Fertilization ⇓ Migration of gravid female to the skin ⇓ Formation of blister ⇓ Ulceration. ⇓ Female discharges embryos in water ⇓ Ingestion of embryos by the cyclops
Manifestations	1. Allergic reactions 2. Cutaneous blister 3. Local intense pruritus 4. Nausea 5. Vomiting 6. Diarrhea 7. Giddiness

	Guinea worm, Serpent worm, Dragon worm, Dracunculus Medinensis.
	8. Emergence of worm from i) Sole ii) Ankle iii) Shoulder iv) Breast v) Testes vi) Knees vii) Tongue viii) Joints
Diagnosis	1. Adult worm comes out like a thread from the centre of the ulcer and can be wound on a stick. 2. Detection of embryos in fluid procured by bathing the ulcer with fluid. 3. Intradermal test: Positive test is indicated by a wheal appearing within 24 hours, if dracunculus antigen is injected intradermally. 4. X-ray: Dead calcified worm 5. Blood examination: Eosinophilia.
Prevention	1. Prevention of infected persons from stepping into source of water. 2. Converting step wells into draw wells, sanitary wells. 3. Filtering water 4. Boiling water
Treatment	1. Niridazole 2. Hetrazan 3. Metronidazole **Removal of worm :** Worm is tied on a match stick and slowly removed over period of days.

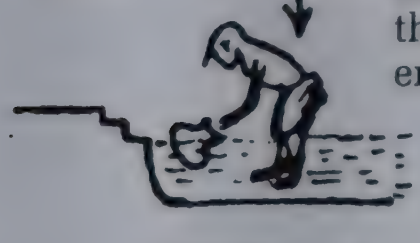
Dracunculus medinensis

Dracunculus medinensis



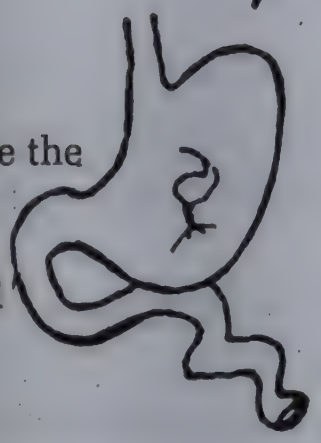
Guinea worm blister

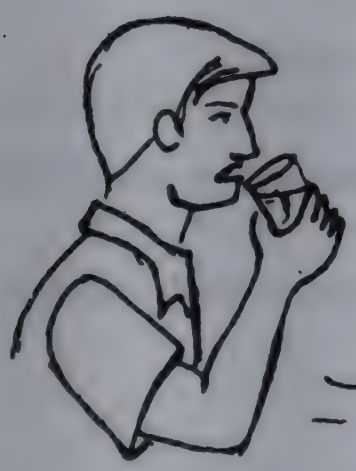
Gravid Female causing blister at the site of emergence



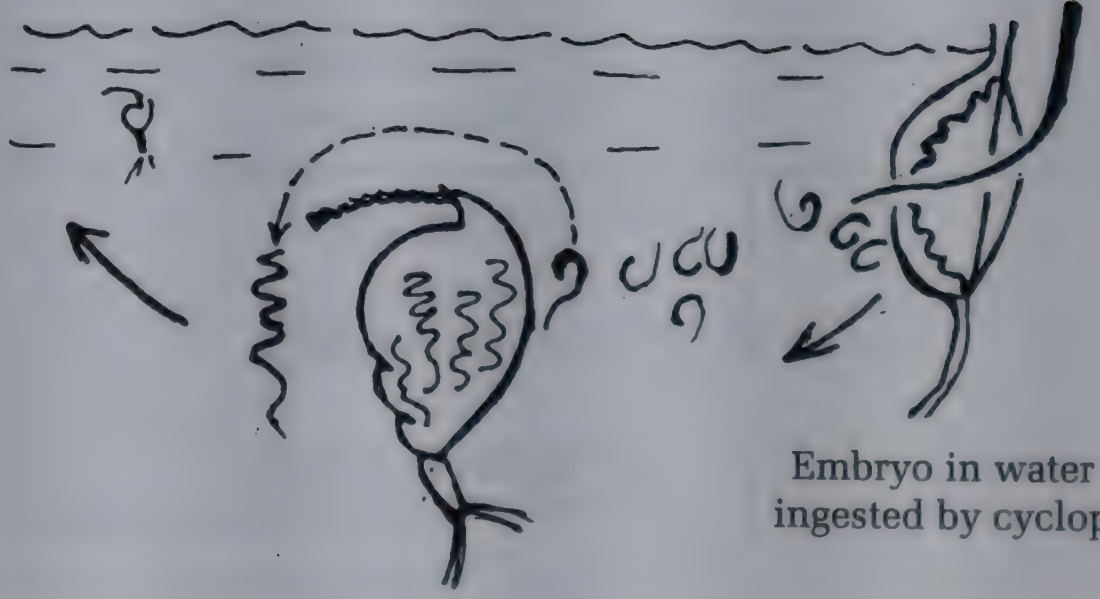
Infection of water supply by patient

Larve penetrate the gut wall grow to maturity in retroperitoneal connective tissue





Human infection from water infected by ruptured cyclop



Embryo in water ingested by cyclop

Development of embryo within cyclop

Filarial worm

Biological name — Wuchereria bancrofti

Mode of entry — Through mosquito bite (Female Culex Mosquito)

Life cycle

Development in man

Deposition of infcctive larvae in the punctured skin
⇓
Migration and settlement of larvae in lymphatics
⇓
Growth and sexual maturity in lymphatics.
⇓
Fertilization
⇓
Gravid female lets off microfilarae
⇓
Entry of microfilarriae into peripheral circulation

Development in mosquito

Ingestion of sheathed micro filariae by mosquito
⇓
Casting off sheath in proventricularis
⇓
Penetration into mosquito's intestinal wall
⇓
Reaching mosquito's thoracic muscle.
⇓
Transformation into first stage larva.
⇓
Second stage larva
⇓
Infective larva
⇓
Entry into mosquito's proboscis ready for injection.

Reproduction — Sexually in lymphatics and liberate microfilariae which enter the thoracic duct. →
→ venous system → pulmonary capillaries → Peripheral circulation

Manifestations

1. Lymphangitis
   a. Upper Limb
   b. Lower limb
   c. Epididymo-orchitis
   d. Funiculitis
2. Lymphadenitis
   Inflamation of regional lymphnodes groin or axilla. Soft lobulated non-tender lymphnodes.
3. Elephantiasis .
   Occurs due to obstruction of lymphatics, due to dead worms. It may affect the extremities or scortum.
4. Hydrocoele.
5. Chyluria.

Filarial worm

Diagnosis

1. Direct Method:
   Detection of adult worm in the lymph node biopsy and calcified worm in x-ray.
2. Indirect method:
   a. Allergic tests
   b. Immunologic tests
   c. Xenodiagnosis (By dissection of mosquito)
      i.e. demonstration of the microfilaria in the mosquito.

Prevention

1. Hetrazan
2. Protection against mosquito bite
3. Destruction of mosquitoes

Treatment

1. Adult worms	:	Synthetic arsenicals, Mel W. Antimonials, MSb B.
2. Microfilariae	:	Diethylcarbamazine citrate Ivermectin.
3. Infective larvae and immature adult worms	:	Paramelaminyl phenyll sibonate (MSb)

10. Presentation of Health Information

All health related data is usually presented in Tables or Graphs or Figures, if there is a need for highlighting certain information.

The methods that are most frequently for data presentation are :

1. Tables.
2. Graphs.
3. Histograms.
4. Bar charts.
5. Pie Charts (sector diagrams)
6. Scatter diagrams.

1. Tables :

If the data is large then the observation are usually grouped into frequency distribution tables.

Example : In a set of 100 observations of cases of Tuberculosis admitted to a hospital. The age wise distributions of the cases can be presented in table form as shown in figure a.

Figure A.

Age	No. of cases
0 — 5 yrs.	18
6 — 15 yrs.	22
16 — 25 yrs.	24
26 — 50 yrs.	18
51 — 60 yrs.	9
61 - 75 yrs.	8
above 75 yrs.	1
Total	**100**

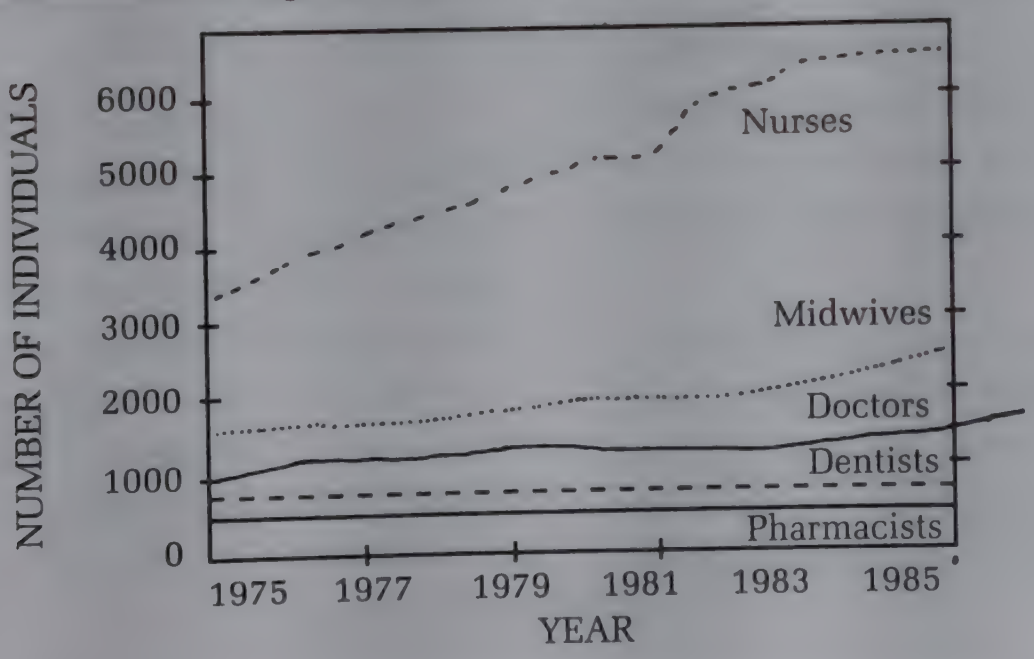
2. Graphs :

These are the most commonly used type of figures particularly for showing numerical data such as deliveries per month, percentage of children immunized by year or number of new cases per month of a disease, such as trypanosomiasis or kala-azar.

Figure b. shows a graph of time trends of the total number of different health workers over a 10 year period. These show that the number of nurses has increased most, from less than 3000 to nearly 6000, with midwives and doctors next. Note, however, that in percentage terms, the pharmacists have increased fastest, showing a nearly 3-fold growth from about 100 to nearly 300.

Graphs are useful for showing two or more distributions, providing the difference between the lines is clearly shown. Figure c. shows the number of mothers delivered each month in a district by trained birth attendants (THA's) during one year compared to the number delivered by professional midwives in the district health centres and hospitals. Assuming that there were a total of 9000 deliveries per year in the district (see Section 3.3), the THA's supervised about 3000 or 33% and midwives a further 1900 or 21% of all deliveries between them; thus, 54% of all births were attended by a trained health worker. The graph also shows that the number of deliveries undertaken by midwives each month rose towards the end of the year, whereas the number undertaken by TBAs remained fairly constant.

Figure B. Growth in number of registered health workers, 1975 - 1985

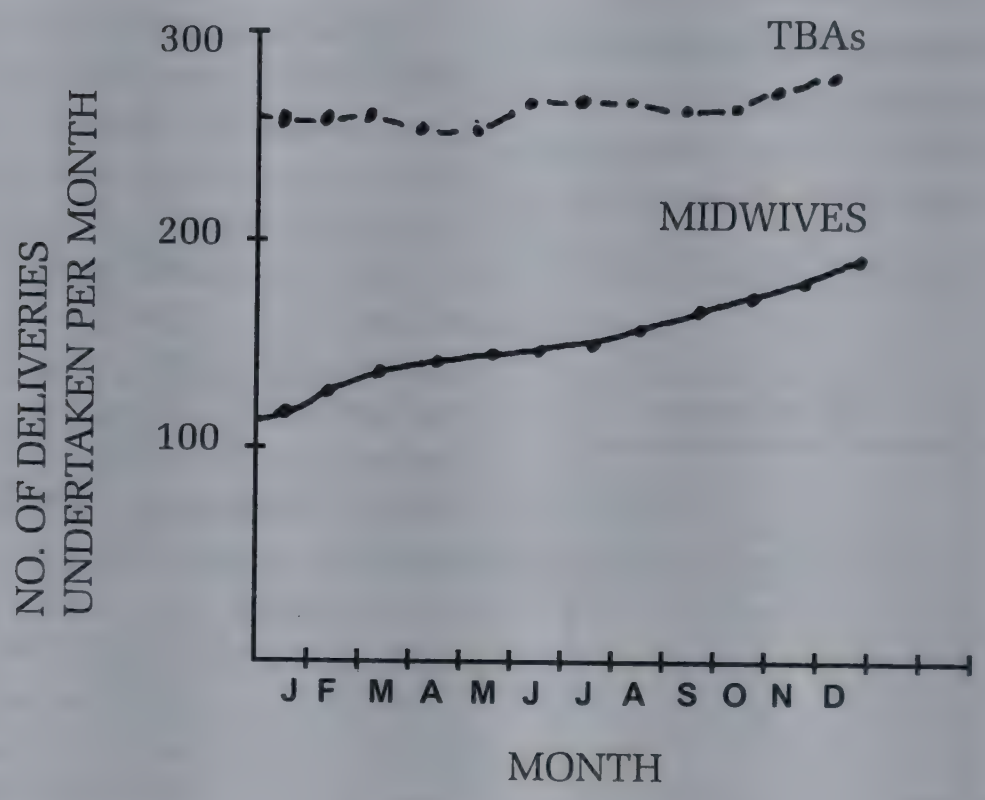


How to draw a straight-line frequency graph

1. Draw the horizontal axis (the X-axis). Mark off the scale using equal units. Use the mid-point of each interval to represent all measurements lying within that interval.
2. Mark off the vertical axis to show the frequency, commonly as a number, percentage or rate.
3. For each class of the grouped data mark a point where the vertical (frequency) and the horizontal (scale) values intersect.
4. Join the marked points with straight lines. The lines can be extended beyond the first and last classes to touch the X-axis. This kind of graph is called a frequency polygon; an example is shown in Figure. d.

Graphs can be used effectively to compare two frequency distributions, e.g. birth weight by sex. Figure d for example, suggests that there were more low-birth-weight female than male babies.

Figure C. Number of reported deliveries undertaken each month by trained birth attendants (TBA's) in homes and by nurse midwives in health centres and hospitals during 1986.



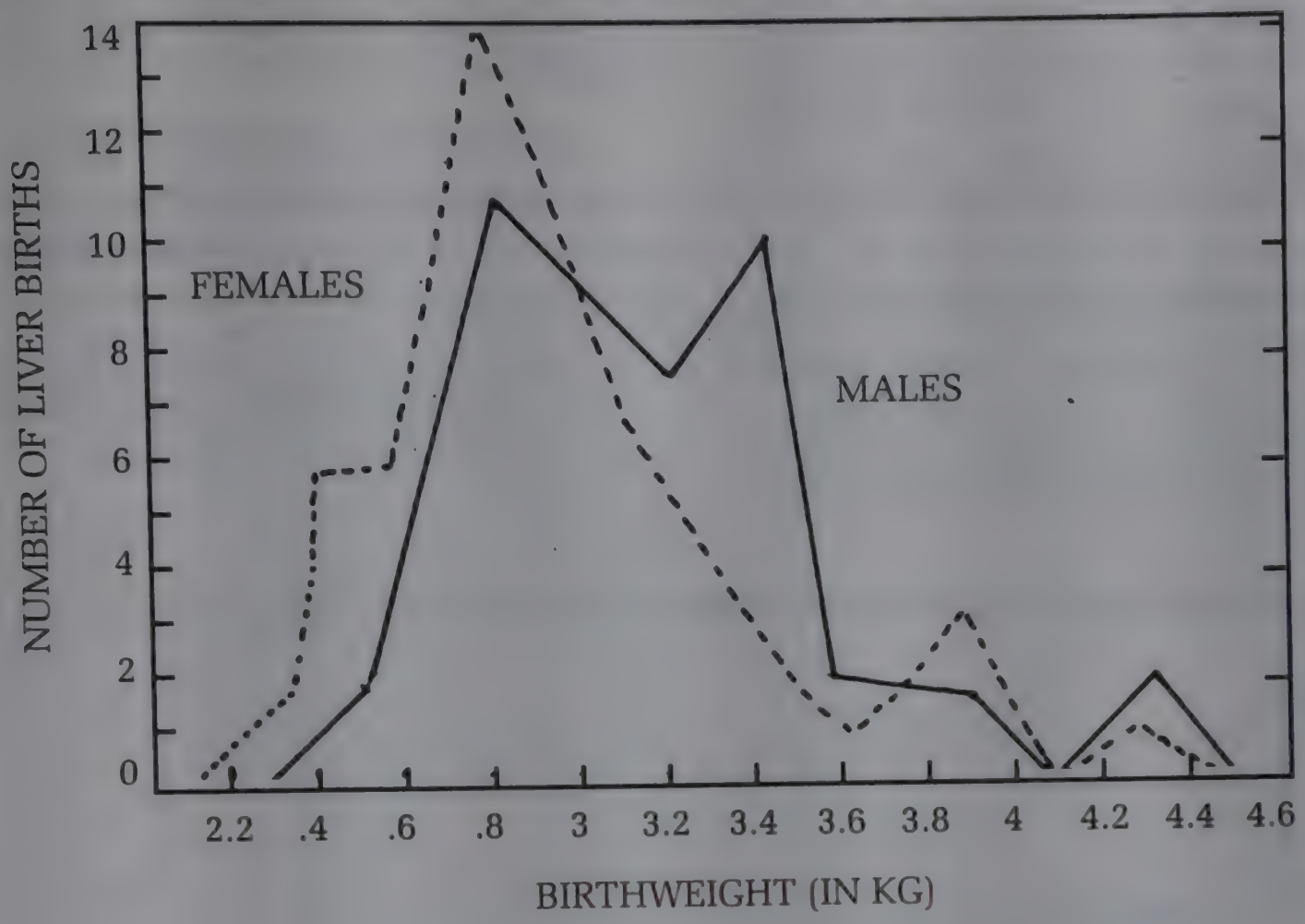
Cumulative frequency graph

This graph expresses a cumulative distribution, often expressed as total numbers or as a percentage. This is a useful graph for showing progress in implementing a planned activity, such as immunization.

Since the frequencies are progressively accumulated, i.e. they always increase (or at least remain constant) over time, the cumulative frequency graph never dips downwards. If no occurrences are addcd to the cumulative frcquency over a time interval, the graph line merely flattens out to give a plateau effect.

An example is given in Figure e. Suppose a district has 40 community health workers (1 per 5000 people) and the Medical officer has decided that they hould be visited once every two months, making 20 visits to be undertaken per month and a total of 240 in one year. Figure e. shows that the visits schedule started off well and then slowed down, but then a special effort was made to carry out more visits in order to catch up towards the end of the year.

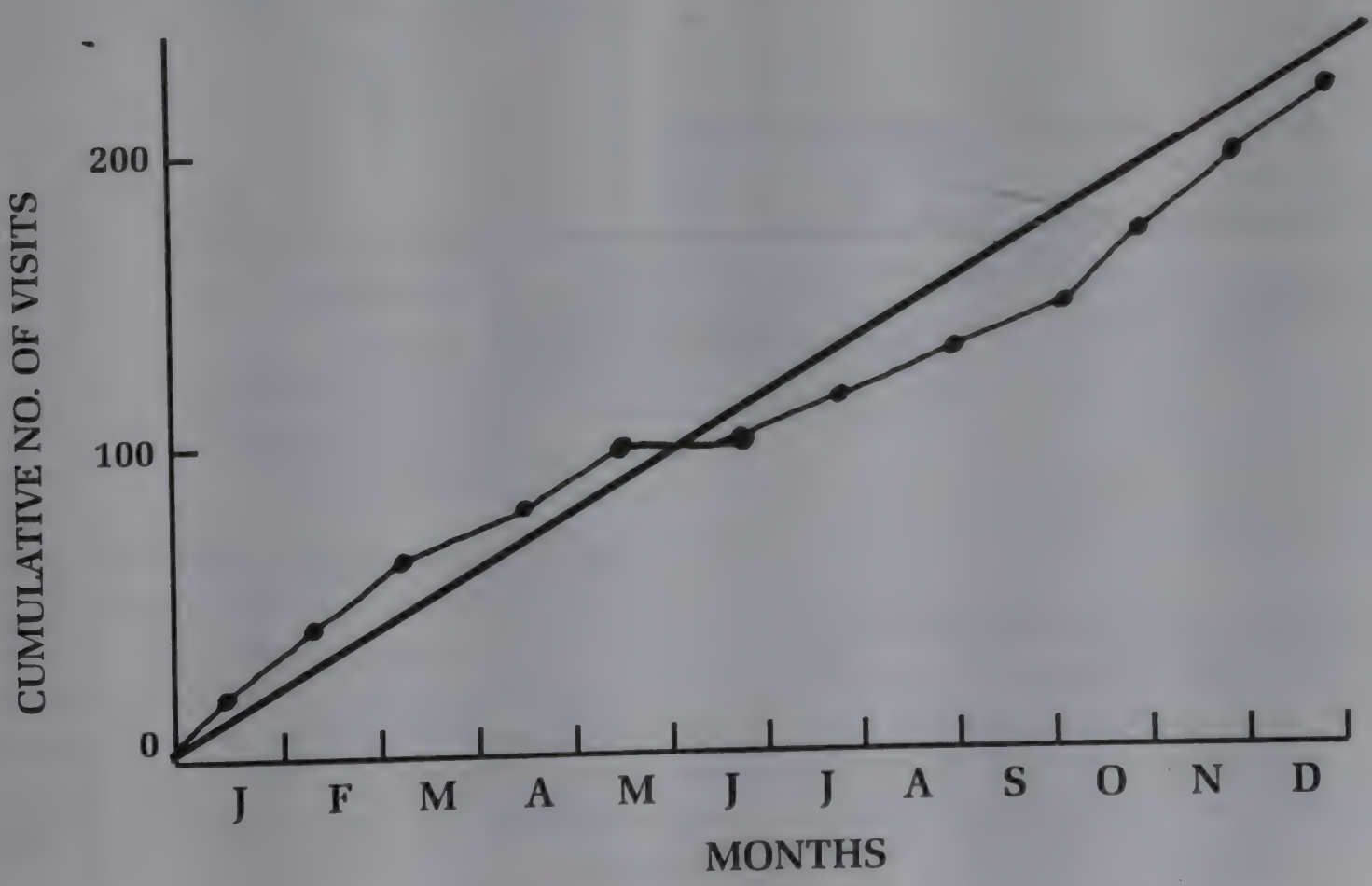
Figure D. Frequency distribution of 100 live births by sex and birth weight



How to draw a cumulative frequency graph

1. Obtain a cumulative frequency distribution by adding the figure in each class of the frequency distribution to all the frequencies in all the preceding classes.
2. For each class, plot the cumulative frequency at the end of the class interval on the horizontal axis.
3. Join the points with straight lines to produce the cumulative frequency graph.

Figure E. Cumulative graph showing number of visit to cummunity health workers by district headquaters staff.



3. Frequency histograms:

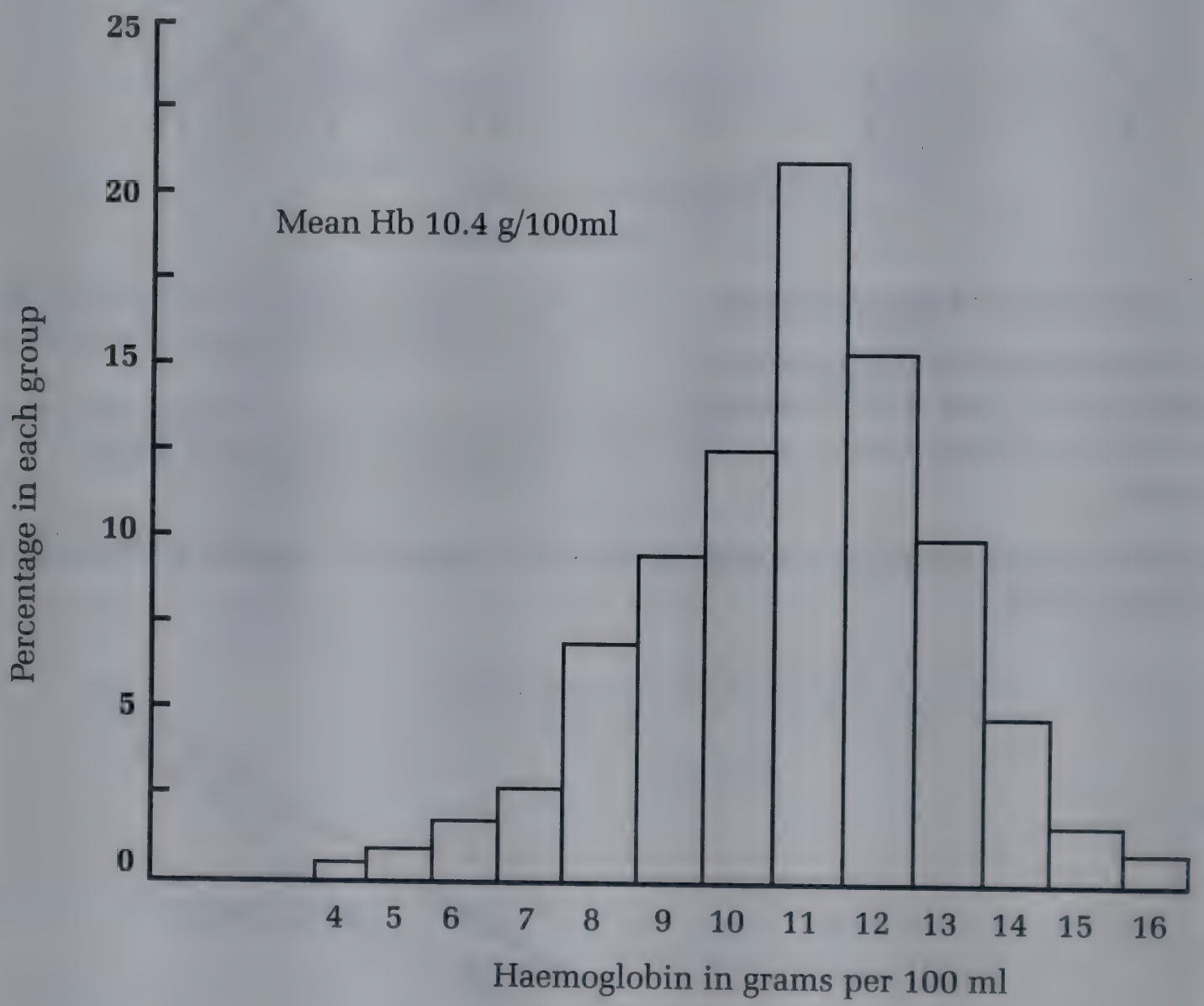
These diagrams are commonly used for presenting information. Figure f. shows a typical frequency histogram. An important characteristic is that the bars of the histogram are contiguous, that is, one bar immediately follows another with no space between. This shows that the scale on the horizontal axis is a continuous measurement scale.

The shape of the distribution in Figure f. is bell-shaped which is the sign of a normal distribution. With this type of distribution it is valid to calculate the mean and the standard deviation for all the individual haemoglobin values. For a one-sided distribution, such as that shown in Figure a. it is valid to calculate the mean, but not the standard deviation.

How to draw a frequency histogram

1. The horizontal axis, the X-axis, gives a continuous scale of the measurement variable while the vertical axis, the Y-axis, shows the frequency.
2. For each class of the grouped data, a bar or rectangle is drawn. The width of the bar is the same as the class interval used.

Figure F. Histogram showing the distribution of haemoglobin levels for 1400 adult men and women.



4. Bar charts :

These resemble the frequency histograms in appearance, but they differ because the bars are not joined together, but separated by a space. This diagrammatic arrangemcnt is used when the horizontal axis deals with information that is qualitative or non- continuous in nature.

Figure g. shows a simple bar chart. It is usual to have the variable or attribute on the horizontal axis and the frequency on the vertical axis. When percentages are used, the sum of the heights of all the bars should be equal to 100%. Occasionally, a bar chart is drawn in which the frequency is represented on the horizontal axis, as shown in Figure h.

When two or more distributions involving descriptive variables need to be compared, a multiple bar chart gives a visual comparison of the two. Figure i. compares the distribution of registered health workers in government and private practice. Each group of workers is represented by a pair of bars, one showing workers in government service and the other those in private practice.

Figure G. Bar chart showing distribution of married couples practising contraception by the main method used.

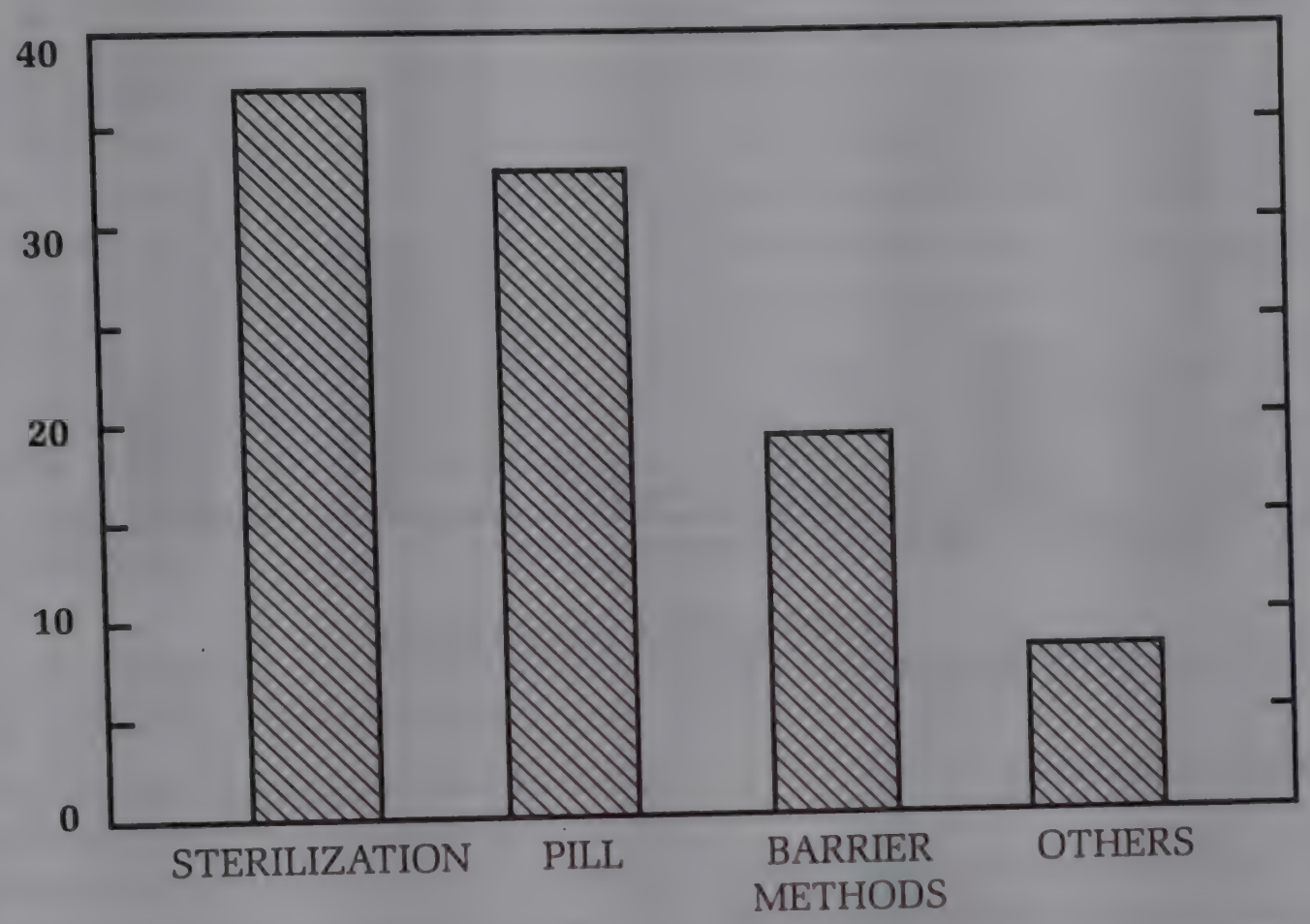


Figure H. Bar chart showing the main source of information on health matters, as reported by individuals in a household survey.

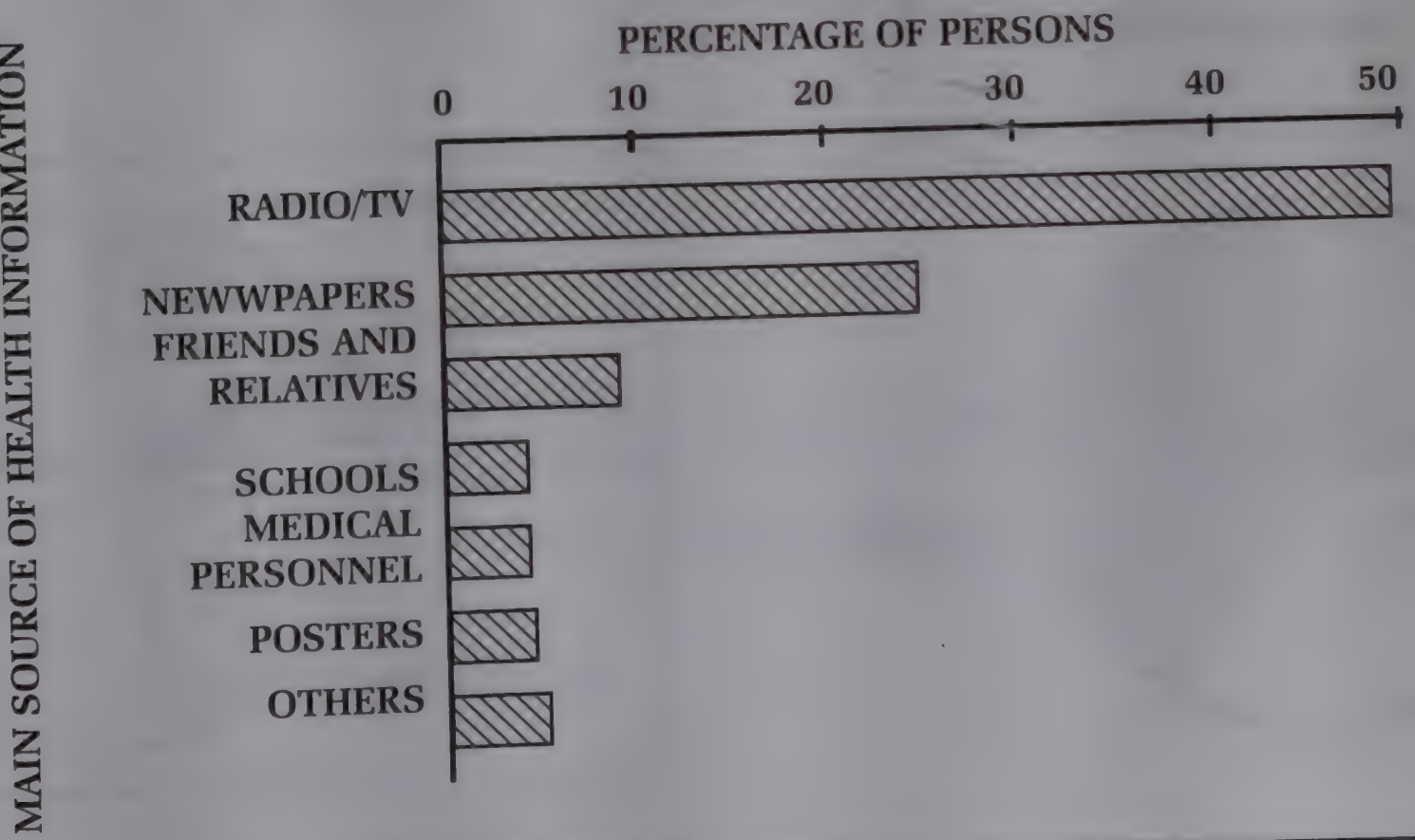
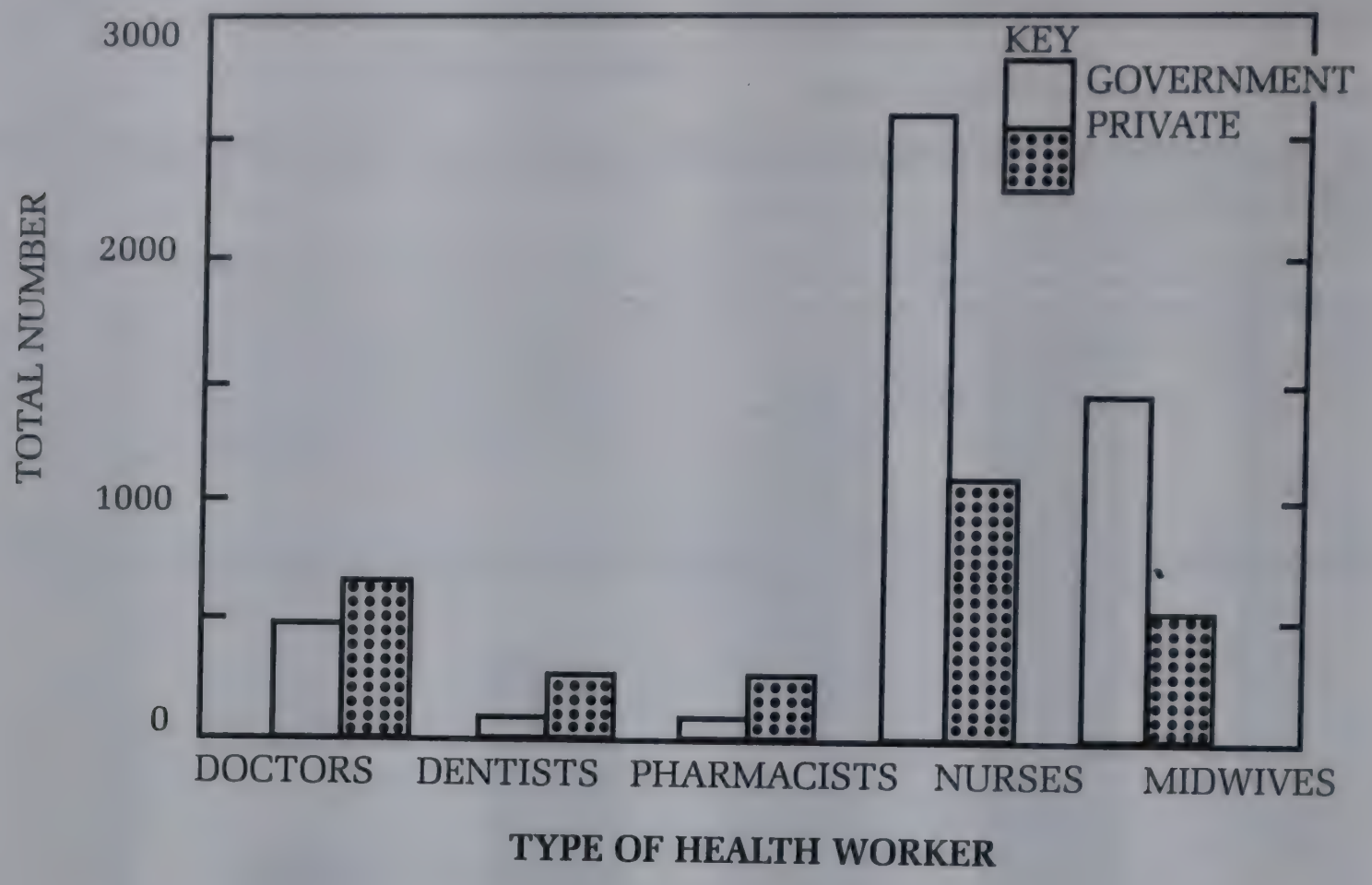


Figure I. Multiple bar chart showing numbers of registered health workers in government employment and in private practice in December 1987



5. Pie charts: (Sector Diagrams)

Figures j. and k. show typical pie charts. These are circular diagrams cut up into several segments or pieces, representing the frequency distribution of the various groups or divisions of a descriptive variable. Pie charts often use percentage distributions, so that a hemisphere represents 50% (half of the pie) and a quadrant 25% and so on. To draw a pie diagram requires the use of a compass, and a protractor for marking out the segments.

The pie chart can also be used for comparing two or more distributions. Pie charts are useful for explaining information clearly to people who are not used to handling numbers.

Figure J. Percentage of the district health budget spent on primary health care facilities during one year

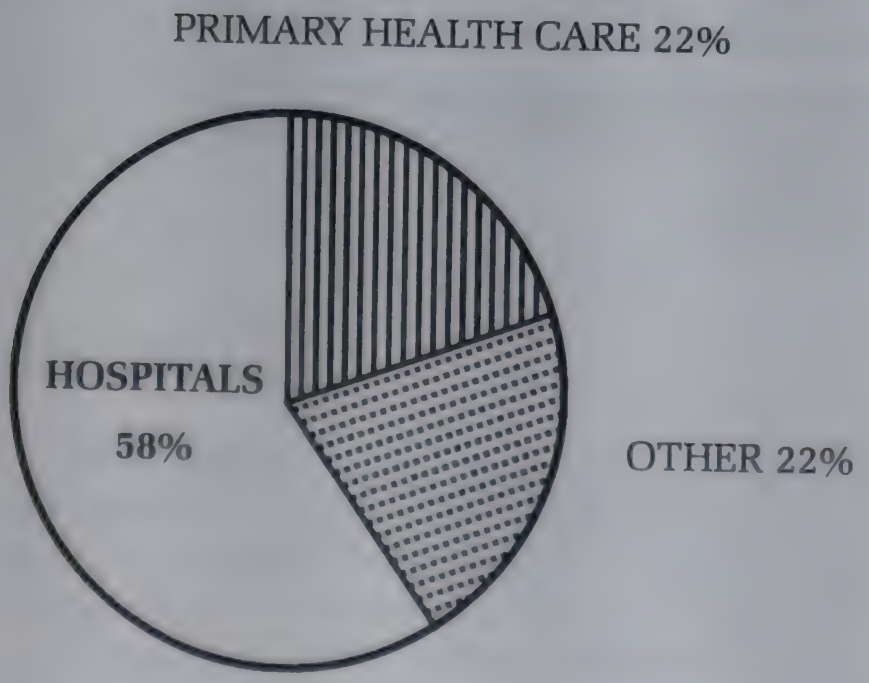
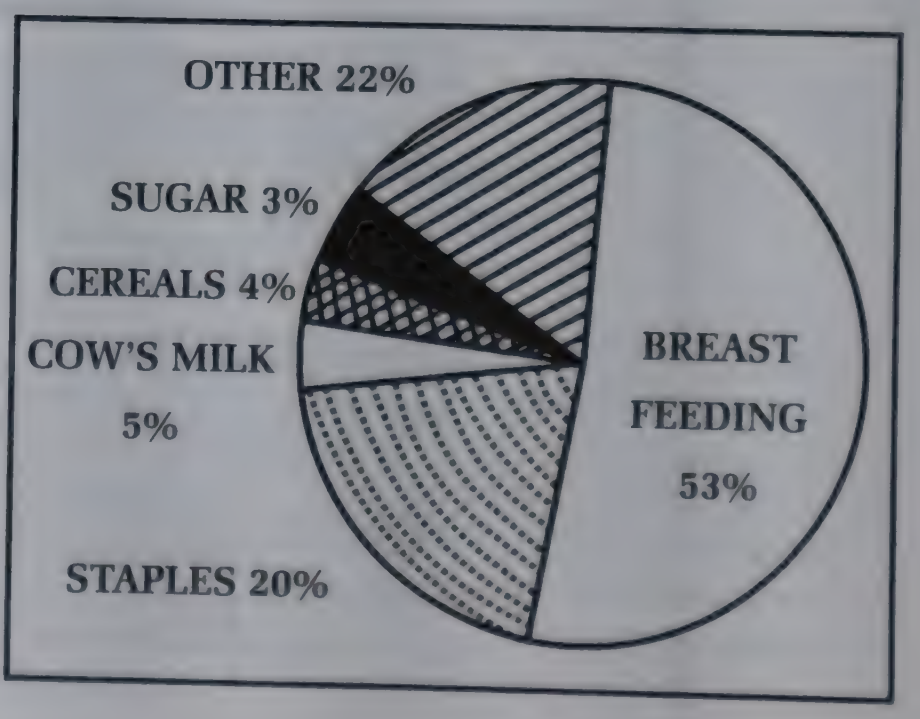


Figure K. Main source of calories for infacts aged 6 to 12 months



6. Scatter diagrams:

These are very useful for displaying information on two connected variables that show a bivariate distribution. For example, when information is obtained on both, the baby's birth weight and gestational age, the two distributions are said to be bivariate.

A scatter diagram is formed when the bivariate distributions are plotted, with birth weight on the vertical axis and gestational age on the horizontal axis (Figure 1.). The name comes from the scatter or spread of the individuals in the sample with respect to the two variables. In drawing the scatter diagram, each dot on the diagram represents the pair of measurements made on one baby. Thus the point marked with a circle in Figure 1. represents an infant whose gestational age was 35 weeks and whose birth weight was 3kg.

Scatter diagrams are used because they show visually whether an association or correlation exists between the two variables. The example Figure 1. shows that there is a positive association between birth weight and gestational age. An infant with a high gestational age tends to be heavier at birth than an infant with a low gestational age. The scatter diagram can only suggest such an association. Statistical techniques are necessary to measure and test the actual strength of this correlation.

Figure 1. also shows that 5 infants had a birth weight below 2500g. that is, they were low-birth weight babies. In this sample, therefore, the low birth weight rate is 5 out of 50, or 10%.

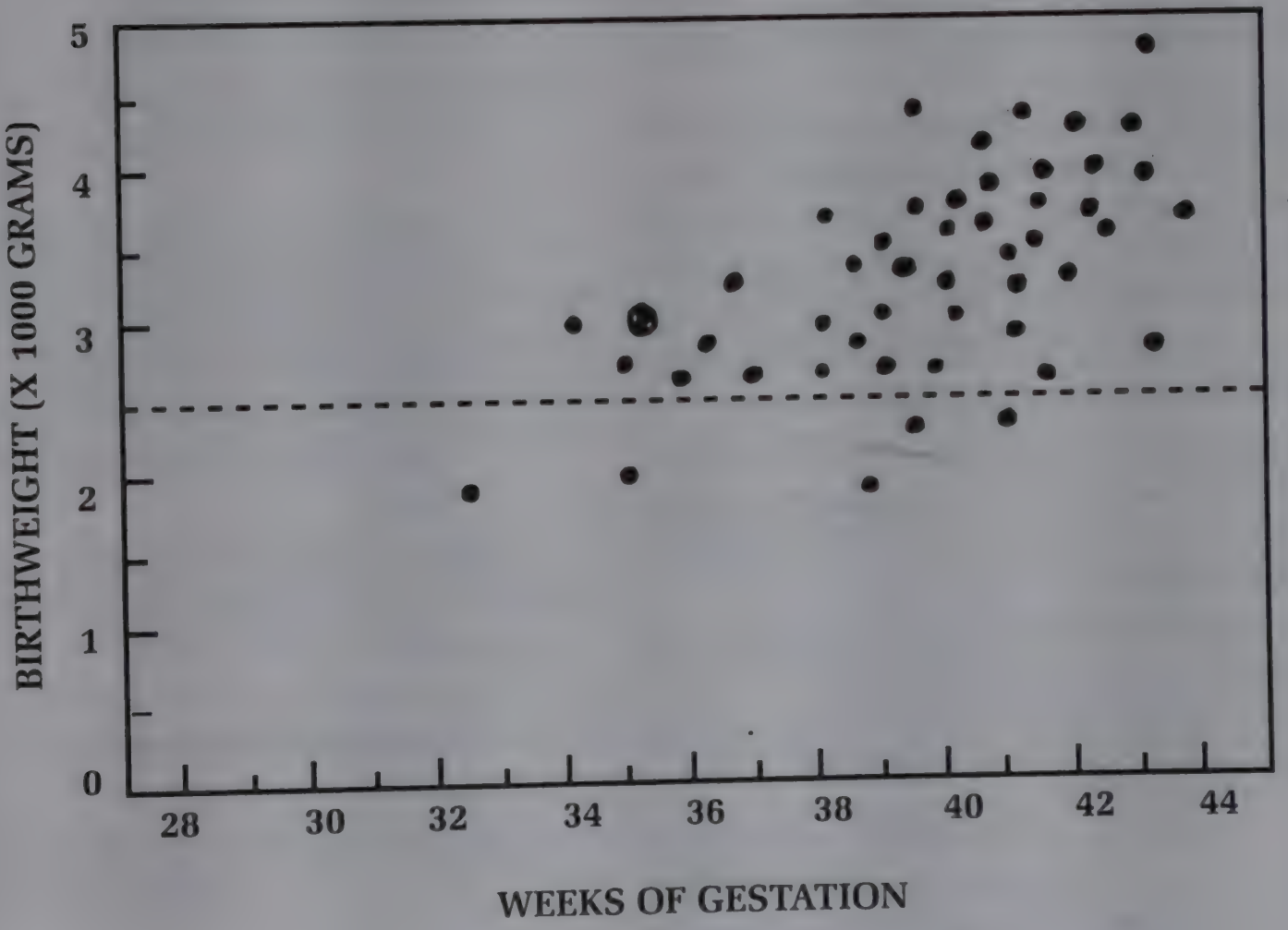


Figure 1. Scatter diagram showing the distribution of 50 live newborn infacts delivered in a hospital by birth weight and gestational age.

11. Environmental Health

A. Horrock's apparatus :

Is used to determine the chlorine demand of water samples.

It consists of

1. One black cup (200 ml.) with a marking. inside.
2. Six white cups (200 ml.).
3. Seven glass stirring rods.
4. One pipette.
5. Two droppers.
6. Starch-iodide indicator solution.
7. Bleaching powder.
8. Two metal spoons which can hold 2gm of , the bleaching powder in each.
9. Instructions booklet.
10. Plastic or wooden box container for all these tems.

Procedure of determining the chlorine demand

1. Take 2g. of bleaching powder in the black cup, add little water and make it into a smooth paste, add more water (upto the mark) stir the solution, allow to settle for 10 mins. This is the chlorine solution.
2. Mark and fill all the six white cups upto the brim with the water to be tested.
3. Using the pipette add one drop of the chlorine solution to the 1st white cup,two drops to the 2nd, three drops to the 3rd and so on till the 6th cup.
4. Mix the chlorine drops in each white cup using a separate stirrer for each.
5. Allow a contact period of 1/2 hr. for the action of chlorine.
6. Add 3 drops of starch-iodide indicator to each of the white cups and stir well (again using the same, separate stirrers, it may be useful to mark or number the stirrers).
7. Presence of excess chlorine (beyond break point) is indicated by the development of blue colour.
8. Note the cups which are showing blue colour. If the 4th, 5th and 6th cup show blue colour it means that excess chlorine is detected from the 4th cup onwards.
9. As per the standardization of the equipment used commonly i.e. the one available from Refrigeration Engineers, Pune if the 4th cup is the first to show blue colouration then 4x2=8 gms. of the bleaching powder is required for disinfection of 455 lines of the tested water sample.
10. To find out the volume of water in a well we have to use the formula

$$V = \pi r^2 h$$

$\therefore$ V = volume
r = radius of the well.
h = height of the water column.

11. There after $\left(\frac{8 \times V}{455}\right)$ will give the amount of bleaching powder required for the well from which the water sample was drawn.
12. There after, disinfection of the well can be carried out by adding $\left(\frac{8\ V}{455}\right)$ gms. of bleaching powder in a metal bucket, (stir well and allow it to settle for 10mins.) The supernatant liquid (chlorine solution) is transferred to another bucket, which is then immersed in the well and moved around vigorously to allow the chlorine to mix with water.

Allow a contact period of 1hr. and later the water from the well can be used for domestic purposes.

B. Chloroscope/Chlorinometer

Is used to detennine the amount of excess/ residual chlorine in water after chlorination.

Consists of

1. Orthotoludine reagent
2. Standard test tubes or discs.
3. Empty test tube.
4. Plastic or metal container.

Procedure

1. Take the water to be tested and add it uptothe mark in the empty test tube.
2. Add drops of O.T reagent 1 :10 parts of water.
3. Shake slowly and match the yellow colour formed with the standard tubes/discs. This gives, directly the amount of residual chlorine in the water if read quickly, (within 10 seconds of adding the reagent). After 15-20 minutes the colour developed is due to free plus combined chlorine.
4. Orthotoludine arsenite (OT A) test is a modification of the O.T test which avoids the errors due to the presence of other elements like, manganese, iron and nitrites. It also allows the reading of free and combined chlorine separately.

C. Dry and wet bulb thermometer (Hygrometer)

This is used for measuring the relative humidity.

It consists of

1. Ordinary mercury thermometer, which measures the room temperature.
2. Another ordinary mercury thermometer, whose mercury bulb is kept moist by covering it with a muslin cloth, whose end is dipping in a small water container.

Procedure:

1. The continuous evaporation of water through the muslin cloth, causes reduction in the temperature in that thermometer (with a cloth). Hence it shows a lower reading than the other thermometer (with out a cloth).
2. The drier the air, more rapid will be the evaporation and hence lower will be the temperature in that thermometer.
3. The difference in the temperatures of the two thermometers varies inversely with the amount of moisture in the air.
4. The humidity can be read from available charts or slide scale.
5. At 100% humidity, both thermometers will show the same temperature. Since then would be no evaporation.
6. The thermometers should be protected from radiant heat, direct sunlight and rain.

D. Maximum and Minimum thermometer.

This is used for measuring the maximum and minimum temperatures of a place on a given day.

This consists of

1. A mercury thermometer with a constriction at the neck of the mercury bulb.
2. A spirit thermometer with a dumb-bell shaped rider or index.

Procedure :

1. When the temperature rises the mercury in the mercury thermometer expands.
2. When the temperature falls, the mercury cannot fall back into the bulb, due to the constriction, hence the mercury column will remain at the maximum temperature.
3. After taking the maximum reading, the mercury thermometer is shaken briskly to push the mercury back into the bulb (like in a clinical thermometer).
4. When the temperature falls, the spirit in the spirit thermometer contracts and drags the rider down along with it, due to surface tension.
5. When the temperature rises, the spirit expands and runs past the rider, hence the rider remains stuck at the minimum temperature.
6. After taking the minimum temperature reading, the rider can be moved to the surface of the spirit column by gently tapping with fingers.

E. Kata thermometer :

Used for measuring

a. Cooling power of air.
b. Velocity of air.

Consists of

1. Two alcohol thermometers each with a bulb of 1.8 cm diameter and 4 cm length.
2. The bulb of one is covered with a wet muslin cloth. This is known as the wet kata thermometer and the other one is known as the dry kata thermometer.

Procedure

1. The bulbs of both Kata's are immersed in warm water till the temperature rises above 130°F.
2. The bulb of the dry kata is wiped dry.
3. The muslin cloth over the wet kata is moistened.

4. The kata's are suspended in air and the time required for the temperature to fall from 100°F to 95°F is noted for both kata's. This is repeated 4-5 times and the mean of the last 3-4 readings is taken.
5. 'Kata factor' is written on the thermometer or the instruction leaflet.
6. Kata factor divided by the mean cooling time gives the cooling power.
7. Dry kata readings of 6 or more, and wet kata readings of 20 or more, are regarded as an index of thermal comfort.

F. Globe thermometer

Used for measuring the Mean radiant heat.

Consists of

1. A hollow copper globe, 15 cm in diameter, which is coated on its external surface with soot or black paint.
2. A mercury thermometer, is inserted into the globe through an opening, so that the mercury bulb lies exactly at the centre of the globe.

Procedure :

1. The black globe absorbs the radiant heat from the surroundings.
2. The mercury thermometer registers the room temperature plus the radiant heat.
3. The difference between the readings of a globe thermometer and an ordinary thermometer kept side by side is equal to the mean radiant heat.
4. The globe thermometer is also influenced by the air velocity.

Note:

A Wet globe thermometer is a globe thermometer whose globe is covered with a wet black cloth. This thermometer exchanges heat with the surrounding by evaporation, radiation, convection and conduction, like a human being. Hence a Wet globe thermometer is considered to provide the most comprehensive measure of the cooling capacity of the environment and is used extensively to measure the thermal comfort in industries

G. Sling psychrometer

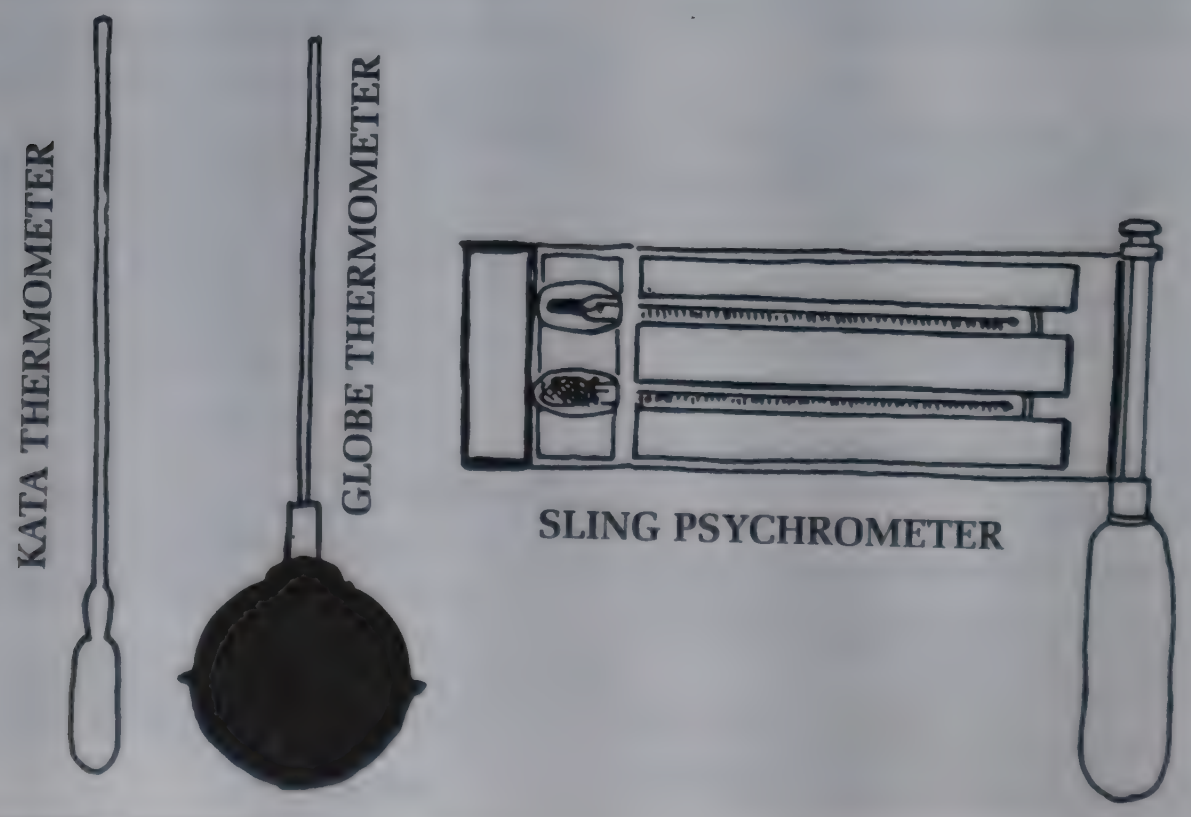
This is used to measure the relative humid

This consists of:

1. One dry and one wet bulb thermometer mounted side by side on a rotating wooden frame.

Procedure :

I. The cloth over the wet bulb is moistened and the wooden frame is rotated for about 15 second at a rate of 4 revolutions per second so as to achieve a rotational speed of 5 meters per second. Note the wet bulb thermometer reading.
2. The psychrometer is again rotated for 10 second and the wet bulb thermometer reading is noted.
3. This is repeated several times, till the wet bulb thermometer temperature remains constant, with no further fall. Now note the dry bulb thermometer reading.
4. Relative humidity can be read directly from available charts.



IV. RECENT TOPICS

1. The Global Eradication of Polio : Target 2000

In May 1988, at its annual meeting in Geneva, the World Health Assembly, the governing body of the World Health Organization (WHO), resolved to eradicate polio from the world by the year 2000. The global eradication of polio involves both halting the incidence of the disease and the worldwide eradication of the poliovirus.

Rationale for Polio eradication : Polio is one of only a limited number of diseases (others include measles and guinea worm disease) that can be currently eradicated. Polio can be eradicated because :

1. Polio only affects humans, and there is no animal reservoir
2. Effective and in-expensive vaccine exists (OPV)
3. Immunity is life-long
4. There are no long-term carriers
5. The virus can survive for only a very short time in the environment.

The polio eradication strategy is based on the premise that polio virus will die out if it deprived of its human host through immunization. The strategy is similar to that used for smallpox eradication in 1977 (In the history of medicine, smallpox is the only disease to have been eradicated so far). Other diseases can be controlled through immunization, but never eradicated. For example, in the case of tetanus, the bacillus that causes the disease (Clostridium tetani) is widespread in the environment and can survive independently from a human host.

The four steps to Polio eradication : The strategy developed by WHO and its member countries to eradicate polio is four aspects:

1. **Routine immunization** with oral polio vaccine(OPV)
2. Additional doses of OPV during **'Pulse Polio-National Immunization Days'**
3. **Surveillance for cases of Acute Flaccid Paralysis(AFP)** and wild polio virus
4. **Mopping-up** immunization activities

1) Routine Immunization with Polio vaccines

Immunity against polio can be stimulated in two ways:

1. Through immunization
2. Following natural infection with polio virus. Polio virus infection provides lifelong immunity against the disease, but this protection is limited to the particular type of polio virus involved (Type 1, 2, or 3). Unfortunately, infection with one type does not protect an individual against infection with the other two types. The development of effective vaccines to prevent paralytic polio was one of the major medical breaktbroughs of the 20th century. Two different kinds of vaccine are available :

1. A live attenuated oral polio vaccine (OPV) developed by Dr. Albert Sabin in 1961.
2. An inactivated polio vaccine (IPV), developed by Dr. Jonas Salk in 1955.

Both vaccines are highly effective against all three types of polio virus. There are, however, significant differences in the way each vaccine works.

Oral polio vaccine (OPV)

Dr, Albert Sabin, developed oral polio vaccine(OPV) in 1961, The OPV acts in two ways:

1. OPV produces antibodies in the blood to all three types of polio virus.
2. OPV also produces a local immune response in the mucous membrane of the intestines, the primary site for polio virus multiplication. The antibodies limit the multiplication of 'wild' (naturally occurring) virus inside the gut, preventing effective infection. This intestinal immune response to OPV is probably the main reason why mass campaigns with OPV can rapidly stop person-to-person transmission of wild polio virus.

Advantages of oral polio vaccine : OPV is an orally administered vaccine and does not require sterile injection equipment. Therefore, trained health workers are not required for it's administration. It can be easily administered by volunteers. The vaccine is relatively inexpensive (current price for-public health programmes in

developing countries is **8 US cents a dose**), which is a major consideration when governments have to purchase massive quantities of vaccine for use during Pulse Polio - National Immunization Days.

In areas where hygiene and sanitation are poor, the short-term shedding of vaccine virus in the stools of recently immunized children, can result in the 'passive' immunization of persons within close contact. The ability of OPV to induce intestinal, local immunity is probably responsible for the effectiveness of OPV mass campaigns in interrupting wild polio virus transmission. Due to these advantages, OPV remains the vaccine of choice for the eradication of polio.

Disadvantages of oral polio vaccine : Although OPV is safe and effective, in extremely rare cases (approximately 1 in every 3 million doses of the vaccine) the live attenuated vaccine virus in OPV can cause paralysis, either in the vaccinated child, or in a close contact. Immune deficiency of the recipient may be among the important causes for this response. This extremely low risk of vaccine-associated polio (VAPP) is well known to, and accepted by most public health programmes in the world because without OPV, hundreds of thousands of children would be crippled every year. Immunization programmes in countries where the risk of wild-virus caused polio has come down to zero are now considering combined immunization schedules using both OPV and IPV.

Inactivated polio vaccine (IPV) :

Dr. Jonas Salk developed IPV(inactivated polio vaccine) in 1955. IPV has to be injected and works by producing protective antibodies in the blood (serum immunity), thus preventing the spread of polio virus to the central nervous system. However, it induces only very low levels of immunity to polio virus locally, in the intestine. As a result, it provides individual protection against polio paralysis, but it cannot prevent the spread of wild polio virus.

Advantages of inactivated polio vaccine:

IPV is not a live vaccine and immunization with IPV carries no risk of vaccine-associated polio paralysis. Immunization with IPV triggers an excellent response of the immune system in most IPV recipients.

Disadvantages of inactivated polio vaccine :

Unlike the oral vaccine, IPV confers only very little immunity in the intestines. When a person immunized with IPV is infected with wild polio virus, the virus can still multiply inside the intestines and be shed in stools (although the vaccinated individual is protected from the disease he will continue to transmit it to others in areas of poor sanitation). For this reason, OPV is the vaccine of choice wherever a polio outbreak needs to be controled, even in countries which rely exclusively on IPV for their routine immunization programme (There was a polio outbreak in 1992 in the Netherlands where only IPV was being given as a part of the national immunization programme). Other disadvantages of IPV include the price (over five times that of OPV), the cost of the syringe, and the need for trained health workers to administer the vaccine using sterile injection procedures.

2) Pulse Polio-National Immunization Days (NIDs)

The second strategy of the four step approach involves mass immunization campaigns, known as Pulse Polio National Immunization Days (NIDs). This supplementary immunization is intended to complement (not replace) routine immunization. The aim of mass campaigns is to interrupt circulation of polio virus by immunizing every child under 5 years of age with two. doses of OPV(regardless of previous immunization status). The objective is to catch children who are either not immunized, or only partially protected, and to boost immunity in those who have been immunized. This way, every child in the most susceptible age group is protected against polio at the same time, instantly depriving the virus of the fertile seed-bed on which its survival depends. NIDs are conducted in two rounds, one month apart (Dec. 6th and Jan. 17th in India). Because OPV does not require a needle and syringe, volunteers with mInimal training can serve as vaccinators, increasing the number of vaccinators well beyond the existing public health staff. The 1997-98 Pulse Polio NIDs in India deployed two million volunteers to immunize 130 million children in just two days. During 1997-98 NIDs, almost 450 million children under five years, i. e. two thirds of the world's children under five years, were immunized.

Three to five years of NIDs are usually required to eradicate polio, but some countries may require more time, especially those where routine immunization coverage is low. NIDs are normally conducted during the cool, dry season because,

immunological response to OPV is improved and the potential damage to heat-sensitive OPV is reduced during .this season (hence the months December and January are chosen for pulse polio immunization in India).

3) Acute Flaccid Paralysis (AFP) Surveillance

The third strategy of the four step approach is surveillance. Surveillance is required to pinpoint where and how wild polio virus is still circulating and also to verify when it has been eradicated.

Effective polio surveillance requires an expert team of virologists, epidemiologists, doctors, and immunization programme staff, backed by good laboratories. WHO, in collaboration with its member countries governments, has established a network of over 100 laboratories (national laboratories, regional reference laboratories, and global specialized laboratories) to provide virological surveillance.

The first link in the chain is the identification of AFP cases. The primary health workers are asked to report promptly every case of acute flaccid (floppy) paralysis in any child under 15 years of age. In addition, primary health workers make regular visits to hospitals and rehabilitation centres to search for AFP cases which were not reported (overlooked or misdiagnosed). Surveillance for AFP by primary health workers is a good example of the '**syndromic approach**' in health care delivery systems. The syndromic approach does not target a specific disease (e. g. polio) but a symptom (acute flaccid paralysis). The rationale of this approach is that it is much easier and more appropriate for the primary health care staff to re.port the symptom of AFP instead of the **'etiological diagnosis'** of polio, and it's importance should be clearly explained to the primary health staff, in order to get their full cooperation in reporting this condition.

WHO emphasizes that all cases of acute flaccid paralysis (AFP) should be reported. The number of cases reported each year is used as an indicator of a country's ability to detect polio. A country's surveillance system should be sensitive enough to detect at least 1 case of AFP for every 1 lakh children under 15 years. For example, in a country of 20 million total population (around 8 million will be children under 15 years) only about 80 cases of AFP are expected per year.

Clinical diagnosis of polio : In the early stages, polio may be difficult to differentiate from other forms of acute flaccid (floppy) paralysis, such as Guillain-Barre Syndrome, transverse myelitis, or traumatic neuritis. Polio virus belongs to the family of enteroviruses, and infections with other enteroviruses can also cause (temporary) acute onset flaccid paralysis. ' Because. of the initial similarity with polio, all patients with the symptom of Acute Flaccid Paralysis, regardless of the initial clinical impression, must be reported and subjected to virological examination, even if the treating physicians are confident on clinical grounds that the case is not polio.

Laboratory analysis : In all reported cases of AFP, to exclude the possibility of polio, faecal specimens are to be obtained and tested for the presence of polio virus. Because shedding of the virus is variable, two specimens, taken 24-48 hours apart, are required for analysis. Speed is essential, since; the highest concentrations of polio virus in the stools of infected individuals are found during the first two weeks after onset of paralysis. Stool specimens have to be carefully sealed in containers and. stored immediately inside a refrigerator or packed between frozen ice packs at 4-8°C in a cold box, ready for shipment. Prolonged exposure to heat on the way to the laboratory may destroy the virus. Specimens should arrive at, the laboratory within 72 hours of collection. Otherwise they must be frozen and transported at -200°C, packed with dry ice. This procedure is, known as the "**reverse cold chain**". At the laboratory, specimens are inoculated into a cell culture. If viruses grow in the cell culture, polio virus must be differentiated from other enteroviruses. Antibodies specific to, individual viruses are introduced to block the growth of these viruses, enabling virologists to single out one of three different sub-types of polio virus.

Mapping out viruses : If polio virus is isolated, the next step is to distinguish between wild (naturaly occurring) and vaccine-related polio virus. This is necessary because the oral vaccine in fact consists of attenuated live polio viruses, and resembles wild virus in the laboratory. They are differentiated by either an ELISA test or a PCR (polymerase chain reaction) test. Once wild polio virus has been identified, further tests are carried out to determine how closely related it is to other strains of wild polio virus and where the identified strain may have originated. By detetrmining the exact genetic make-up of the virus, wild viruses can be classified into genetic families which are

known to cluster in specific geographical areas. The virus sequence is then checked against a reference trains of known polio viruses, which gives clues about the geographical origin of the virus. When the polio virus has been pinpointed to a precise geographical area, it is possible to identify the source of origin of the polio virus. Appropriate immunization strategies can then be determined to avoid further spread of the polio virus. Virologists have shown that mutations occur in the genetic material of a polio virus each time it is transmitted trom person to person. Over a year, the genetic make-up of the virus can change by 2%. When differences of greater than 10% are identified between viruses, they are no longer considered to belong to the same genetic family.

4) Mop-up or Mopping-up rounds of immunization for polio eradication

When very few or no cases of polio occur, the final strategy of the four step approach is implemented. This involves door-to-door immunization ("mopping up") in high-risk districts where the virus is known or suspected to be still circulating. Priority districts include those where polio has occurred over the previous three years and where access to health care is difficult. Other criteria include overcrowding, high population mobility, poor sanitation, and low routine immunization coverage.

2. World Health Day

1980	Smoking or Health: The Choice is Yours
1981	Health For All By The Year 2000 AD
1982	Add years to life
1983	Health For All by 2000 AD : The Countdown Has Begun
1984	Children's Health; Tomorrow's Wealth
1985	Health Youth; Our Best Resource
1986	Health Living - Everyone's a Winner
1987	Immunization - A Chance for Every Child
1988	Health for All - All for Health
1989	Let's Talk Health
1990	Our planet - Our Health; Think Globally Act Locally
1991	Should Disaster Strike - Be Prepared
1992	Heart Beat - The Rhythm of Life
1993	Handle life with care - Prevent Violence and Negligence
1994	Oral Health for a Healthy Life
1995	Target 2000-A World without Polio
1996	Healthy Cities for a Better Life
1997	Emerging Infectious Diseases: Global Alert, Global Response
1998	Invest in the Future: Support Safe Motherhood
1999	Active, ageing makes the difference
2000	Safe blood starts with me blood saves lives
2001	Mental health : Stop exclusion dare to care
2002	Move for health

3. Important days in the Health Calendar

January	17th	Pulse Polio Day National Immunization Day)
	30th	Anti Leprosy Day
March	8th	International Women's Day
April	7th	World Health Day
	22nd	World Habitat Day
May	8th	World Red Cross Day
	15th	International Day of Family
	31st	World No - Tobacco Day
June	5th	World Environment Day
	26th	Inlernalional Day againsl Drug Abuse and Illicil Trafficking.
July	1st	Doctor's Day
	11th	World Population Day
August	1st	World Breast - feeding Day
	1st to 7th	World Breast-feeding Week
	20th	World Mosquito Day
September	1st to 7th	National Nutrition Week
October	8th	World Elders Day
	9th	World Breast Cancer Awareness day
	11th	National No Tobacco Day
	13th	International Day for Natural Disaster Reduction
	16th	World Food Day
November	15th	World Diabetes Day
	18th	World Epilepsy Day
December	1st	World AIDS Day
	2nd	National Pollution Prevention Day
	6th	Pulse Polio Day National immunization Day
	8th	International Day of the Handicapped
	11th	UNICEF Day

4. Child Labour & International Labour Organisation

Child labour includes, children prematurely leading adult lives, working long hours for low wages under conditions damaging to their health and to their physical and mental development, sometimes separated from their families, frequently deprived of meaningful educational and training opportunities that could open up for them a better future. Age is obviously the most important criteria for distinguishing child labour from adlult labour. The **definition of a child** in terms of age differs according various Acts. Factories Act prohibits children below the age of 14 from working in any factory. The limit in the Mines Act is 15 years and it is 12 the Plantation Labour Act. Article 24 of the Constitution of India states "No child below the age of 14 years shall be employed to work in any factory or mine or engaged in any other hazardous employment". The most recent "The Child Labour (Prohibition and Regulation) Act, 1986 defined clild as a person who has not completed his fourteenth year of age.

Manitude of the problem

Millions of child workers are employed around the world in conditions dificult or dangerous for their health. India has a average of 25% of children between the ages of five and fifteen are in full or part time jobs. Working is unusual among children under ten but increases rapidly in the 10 - 14 yrs. age group. The largest group are children who worked without pay within their own family. All India Child Labour Sample Survey conducted by the Operations Research Group in 1980-81 revealed that ere are 44 million child workers in India. This is about a fourth of the world's child labourers. In India, Child workers formed about 7% of the total work force. In 1975, the International Labour Organization had singled out India as having the largest number of child workers and that too in the age group of 10-14 years alone.

The existence of child labour in whatsoever magnitude should be a matter of concern for the working children, the parents, society and the State. Whether part time or full time, in any occupation, engagement of a child in the labour force itself means a complete or partial denial of childhood to him. He is not merely deprived of the joys and carefree life of child but also for a desirable physical and mental development. This is not only injustice to him as a child but also as an adult throughout his life, for the foundation of his adulthood is built on extremely weak structure of uderdevelopment.

Most of the working children come from very poor families and supplement the family earnings. This is natural in a country where almost half of the population lives below even the official poverty line. About 85% of working children belong to the rural areas and are employed in agricultural and allied activities in the rural sector. On an average, children earn about 50% of what adult men earn. Thus making a significant contribution to total family's earnings. In the urban sector, more than 1/3rd of the total work force are children and of that more than one fourth belong to the non household industries and construction.

A committee on Child Labour constituted in 1979 has observed that child workers in India are commonly employed in the following occupations: Agriculture, Plantations, Mining & Quarrying, Beedi Making, Glass & Bangles Making, Handloom & carpet weaving, Zari & Embroidery Work, Gem Cutting & Polishing, Match Boxes & Fireworks Making, Machine Tools, Repair shops, Petrol pumps, Cashew nut Processing, Manufacturing of Coir products, Domestic workers, Helpers in Hotels, Restaurants, Canteens, Tea stalls, Shops & Way- side Establishments, Rag-picking, Construction, Hawkers, vendors, Newspaper sellers, Coolies etc.

The problems associated with child labour

1. **Exploitative Working Conditions:** The children are :

a) Engaged in work that jeopardizes their physical and mental health & well being. They are forced to do hazardous work in unsafe working conditions. There is danger to the child's life and health because of their employment in hazardous jobs, or by the unsafe working environment. Such dangers are further compounded by fatigue due to excessive hours of work, under nutrition and the physical weakness of children. Certain occupations are classified as hazardous. But as far as a child work is concerned, every occupation is

hazardous for his well being, growth and development. The health hazards in some of the occupations where child workers are employed are summarized here:

1) Beedi industry	Chronic bronchitis and Tuberculosis.
2) Glass industry	Asthma, Bronchitis, Tuberculosis, Eye disorders.
3) Handloom industry disorders.	Asthma, Tuberculosis.
4) Zari and Embroidery	Eye disorders.
5) Gas cutting and diamond cutting	Eye disorders.
6) Rag pickers	Tetanus, Skin diseases.
7) Pottery	Asthma, bronchitis, TB
8) Stone quarries / Slate quarries	Silicosis.

2. Low Remuneration and Excessive Hours of Work :

a) The children are not given adequate rest breaks during the day.

b) No weekly rest and no annual holidays are given.

c) Among the most frequent violations of child labour laws is the working of hours in excess of the number permitted, or working at night beyond the times permitted. The hours of work as per the Minimum Wages Act, 1948 would be 4½ hours for six days in a week. No child shall work at night (between 7 p.m. to 7 a.m.). In actual fact, most of the children are found working for 7 hours or more per day. The regulation the hours of work in relation to children is being violated with impunity by the different industries employing child labour.

d) The remuneration is well below the prescribed minimum wages. The committee of the State Labour Ministers set up in 1983, has pointed out that wages for children on time rate should be on par with wages for adults, and not less than 75% of the wages for the latter in any case. However, the situation in India is that a working child is paid merely Rs. 90 per month.

3. Physical Abuse and other Psychological Problems :

a) Children are traded or contracted out for long periods and are often separated from their parents for very long duration.

b) Children are also beaten and/or starved, sometimes resulting in the death of the child.

c) Denied their right to play, to learn, and in general to enjoy a normal childhood.

Factors Responsible for Child Labour

In developing countries, the problem of child labour in both rural and urban areas is principally one of poverty and unemployment, which are themselves a result of unequal distribution of land and production assets and low levels of development. About 328 million people in India live below the poverty line. Nearly 14 million people are unemployed. In fact, the inevitable result of poverty inequitable distribution of land and assets, unemployment, non payment of minimum wages is BONDED LABOUR. Large numbers of bonded labourers began their lives as child labourers. As children they are mortgaged to the landlord/ money lenders by their parents.

a) Chronic poverty is the fundamental cause for the perpetuation and prevalence of child labor.

b) Non schooling of children is also related to child labour.

c) Inefficiency of protective legislation for working children.

d) No effective inspection system.

e) Employer's preference for child workers is yet another major cause for the prevalence of child labour.

International Labour Organization (ILO) : Conventions / Recommendations

The ILO has paid special attention to the protection of children and young persons. It has sought to achieve its objective of protecting children by adopting international labour standards in the form of Conventions and Recommendations. A Convention seeks to create obligations of a binding nature and its ratification requires complete compliance with all its provisions. A recommendation on the other hand

contains provisions which are generally in the nature of guiding principles for action and may be supplemented progressively. So far there have been 18 conventions in 180 respect to children, these are concerning their minimum age for entry to employment, medical examination and working at night. There are also 9 recommendations on these topics. These are a part of the International Labour Code.

The Child Labour (Prohibition and Regulation) Act (1986)

The act came into force on 23.12.1986. It bans the employment of children in specified occupations & processes.

Prohibited occupations :

1) Transport of passengers, goods or mail by railway.
2) Cinder picking, cleaning of an ash-pit or building operation in the railway premises.
3) Work in a catering establishment at a railway station.
4) Work relating to the construction of railway station or any work in close proximity to or between the railway lines.
5) A port authority within the limits of any ports.

Prohibited processes :

1. Beedi making.
2. Carpet weaving.
3. Cement Manufacturer, including bagging of cement
4. Cloth printing, dyeing, and weaving.
5. Manufacturer of matches, explosives and fireworks.
6. Mica cutting and splitting.
7. Shellac manufacture.
8. Soap manufacture
9. Tarring.
10. Wool cleaning.
11. Building and construction industry.

This Act also specifies the hours and period of work for children:

- No period of continuous work shall exceed 3 hours and a minimum rest period of one hour must follow the three work period.
- The work period of a child shall not be more than 6 hours per day inclusive of one hour rest period.
- No child shall be permitted to work between 7 p.m. and 8 a.m.
- No child shall be permitted to work over time.
- Section 8: States that every employed child shall be allowed every week, one full day holiday.
- Maintenance of a register - This register must maintain all the records regarding children and should be available for inspection by an inspector at all times.
- Notice to Inspector : Every occupier of an establishment who employs or permits a child to work shall send a written notice to the local inspectors of factories and establishments, stating the nature of work done, number of children employed.
- Section 14: deals with penalties
- Imprisonment from one month to 2 years and fines from Rs. 10,000 to Rs 20,000 or both, depending on the offense.

This act emphasizes on penalties as compared to the earlier acts. Thus we can clearly see that there are a number of acts to safeguard the interest of the child worker. Yet just the presence of an Act is not enough. It must enforced. It has been seen over the years that the actual implementation of these acts does not take place. There are various reasons that have been observed in regard to this aspect. First of all there are several inadequacies in one existing administrative set up for the implementation of various laws.

1. The jurisdiction of individual inspectors was too extensive for them to keep a regular watch on the activities within their purview.
2. Some of the posts were vacant. The present requirement is that an inspector must visit 150 factories twice a year but because of their inadequate number, it is impossible to comply with the legal requirement. There are a large number of factories which remain uninspected in a year and some are visited only for the purpose of investigating accidents.
3. Also earlier, the punishment provided as penalty for violation of Acts was very meager and therefore, had no deterrent effect.
4. There is a deliberate attempt by the employers to flout the legal provisions. When an inspector visits a factory or an establishment, the employer sends the children away through the back door.
5. No registers of children are maintained. The practice of keeping duplicate registers is also very common.

6. It is very difficult to get evidence of one pledging of child labour.
7. Also the managements are in possession of dubious age certificates in respect of all children employed by them. In many cases, children who re hardly 8 to 10 years of age, are certified as having completed 15 years of age.

What's the use of laws on paper ?

Inspite of so many legislations on child labour, children continue to forcibly toil under hazardous conditions and the employers are successfully escaping the penalties prescribed under these Acts. An estimated 49 million child workers are currently working in India and 2 million of these children are engaged in hazardous industries. In India, child workers are considered essential to maintain the economic level of the household either in the form of work for wages or in the form of help in household enterprises. Poverty was a key factor leading to child labour and the parents and relatives expected children more often girls to perform domestic chores that feed adult members of the household, as well as, to on an income outside.

Social workers feel that enforcement of free and compulsory education will be more effective than legislation in curbing the practice of child labour. Child labour aggravates unemployment, poverty and exploitation since child workers are paid less than adults. There is a direct correlation between the unemployment level and child labour.

Child labour cannot be abolished for its roots lie in object poverty. Thus unless the social and economic conditions are improved and the children start going to school, we cannot hope to eliminate child labour. We should concentrate on ensuring basic human needs of all the people. They include such essentials as food, shelter, clothing, water, education, training and provision for gainful employment. Child labour is embedded in poverty and it is although sustained increase in standards of living that it will be abolished.

Some measures for the control of child labour (Recommendations of the I.L.O. 1983):

1. The adoption and strict enforcement of laws or regulations prohibiting the employment or work of children in hazardous activities.
2. The promotion of occupational safety and health and the improvement of the physical environment at the work place in sectors where children are known to be engaged.
3. The regulation of work of children in respect of hours of work, night work, rest and weekly holidays.
4. Provision of welfare facilities and services for children at / or near the workplace.
5. The expansion of educational facilities to permit the extension or more effective implementation of compulsory education.
6. The introduction of schemes by which children who are obliged to work can combine remunerative activity with education or training.
7. The encouragement of action by employees trade union and voluntary organization to promote child welfare.
8. The exposure of particularly abusive or exploitative practices.
9. The dissemination of information designed to create greater public awareness of the adverse effects of child labour.

What can we do ?

1) Create general awareness about the evils of child labour.
2) Identify and blacklist the products from industries which employ children.
3) Highlight the problem in national press and through campaigns.
4) Ensure efficacious implementation of the constitutional provisions relating to child labour; the child labour (Prohibition and Regulation) Act, 1986 and the National Child Labour Policy.

Remember, "Children are the most important asset of any nation. Let us offer our children an environment which is full of opportunities for their education, growth and development."

DECLARATION OF THE RIGHTS OF THE CHILD Proclaimed by the United National General Assembly on 20 November 1959 (Resolution 1386) (XIV) to the end that he/she may have a happy childhood and enjoy for his/her own good and for the good of society the rights and freedoms herein set forth, and calls upon parents, upon men and

women as individuals and upon voluntary organizations local authorities and national governments to recognize these rights and strive for their observance by legislative and other measures progressively taken in accordance with the following principles:

Principle 1:

The child shall enjoy all the rights set forth in this Declaration. Every child without any exception whatsoever, shall be entitled to these rights, without intimidation or discrimination on account of race, colour, sex, language, religion, political or other opinion, national or social origin, property, birth or other status, whether of himself or of his family.

Principle 2 :

The child shall enjoy special protection and shall be given opportunities and facilities by law and by other means to enable him to develop physically, mentally, morally, spiritually and socially in a healthy and normal manner and the conditions of laws for this purpose the best interests of the child shall be the paramount consideration.

Principle 3 :

The child shall be entitled from his birth to a name and a nationality.

Principle 4 :

The Child shall enjoy the benefits of social security. He shall be entitled to grow and develop in health and to this end special care and protection shall be provided together to him and to his mother, including adequate prenatal and post natal care. The child shall have the right to adequate nutrition, housing, recreation and medical services.

Principle 5 :

The child who is physically, mentally or socially handicapped shall be given the special treatment, education and care required by his particular condition.

Principle 6 :

The child for the full and harmonious development of his personality, needs love and understanding. He shall wherever possible, grow up in the care and under the responsibility of his parents, and in any case, in an atmosphere of affection and of moral and material security. A child of tender years shall not (other than in exceptional circumstances) be separated from his mother. Society and the public authorities shall have the duty to extend particular care to children without a family and to those without adequate means of support. Payment of state and other assistance towards the maintenance of children of large families is desirable.

Principle 7 :

The child is entitled to receive education, which shall be compulsory at least in the elementary stages. He shall be given an education, which will promote his general culture and enable him, on a basis of equal opportunity to develop his abilities, his individual judgment and his sense of moral and social responsibility and to become a useful member of society. The best interests of the child shall be the guiding principle of those responsible for his education and guidance and that this responsibility lies in the first place with his parents. The child shall have full opportunity for play and recreation, which should be directed, to the same purposes as education. Society and the public authorities shall endeavor to promote the enjoyment of this right.

Principle 8 :

The child shall in all circumstances be among the first to receive protection and relief.

Principle 9 :

The child shall be protected against all forms of neglect, cruelty and exploitation. He shall not be the subjected to trading in any form. The child shall not be admitted to employment before an appropriate minimum age, he shall in no case, he case be permitted to engage in any occupation or employment which would prejudice his health or education or interfere with his physical, mental or moral development.

Principle 10 :

The child shall be protected from practices, which may foster social, religious or any other form of discrimination. He shall be brought up in a spirit of understanding, tolerance, friendship among people peace and universal brotherhood and in full conscious that his energy and talents should be devoted to the service of his fellow men.

5. WHO's New programme - "Roll Back Malaria - A Global Partnership

Introduction

Upon taking office in July 1998, the World Health Organization's new Director-General, Dr Gro Harlem Brundtland, decided that malaria was to be one of WHO's top priorities.

There are an estimated 300-500 million cases of malaria per year. The majority of these occur in Africa, while the vast majority of the estimated 1 million annual deaths from the disease occur among children, and mainly among poor African children. Malaria is above all a disease of the poor, impacting at least three times more greatly on the poor than any other disease. Although malaria had been a priority of WHO since its inception in 1948, malaria control efforts had often suffered from a lack of financial resources and uneven implementation.

RBM team

Roll Back Malaria will be run with a central team of eight to 10 people headquartered in WHO in Geneva. The team will be led by Dr David Nabarro, who until his appointment as RBM project manager was Chief Health Advisor and Strategic Director of the United Kingdom Department for International Development.

Objectives

The goals of RBM will include :

Support to endemic countries in developing their national health systems as a major strategy for controlling malaria ;

Efforts to develop the broader health sector (i.e., all providers of health care to the community - the public sector health system, civil society and non-governmental organis.uions, private health providers [including drug vendors and traditional healers] and others) ;

Encouraging the needed human and financial investments, national and international, for health system development.

RBM's implementation at country level will provideoan indicator of the effectiveness of these health systems, while the programme will also serve as a model for WHO in developing both other global health and development initiatives and new methods of controlling infectious diseases.

RBM: A new approach to malaria control

WHO's partners in RBM will include malaria endemic countries, other UN organisations (on 30 October 1998, the United Nations Development Programme, UNICEF, the World Bank and the World Health Organization announced that the four agencies were launching RBM jointly and that they would cooperate in all aspects of its activities), bilateral development agencies, development banks, non-governmental organisations and the private sector.

WHO's role in the global partnership will be to :

1) Provide strategic direction and catalyse actions
2) Provide an RBM secretariat of approximately eight to 10 people at its Geneva headquarters
3) Work to build and sustain country and global partnerships
4) Arrange the provision of technical endorsement, directly or through approved resource networks, for both a collective strategy and for individual partners' actions
5) Ensure that all aspects of progress of RBM are monitored
6) Provide global accountability for RBM
7) Broker technical assistance and finance on behalf of those who need it
8) Undertake responsible advocacy for the RBM approach to reducing malaria-related suffering

The role of UN partner agencies

UNICEF will

1) Provide support to intensified malaria control efforts via its country programmes.
2) Work with Government & NGO partners to: give special attention to reducing the terrible toll of malaria on young children and pregnant women.

3) Further strengthen support for community-based and local action to improve health and nutrition;
4) Focus on making insecticide treated mosquito nets available to all families that need them and on ensuring that every child with malaria has access to early and effective treatment; mobilize leaders (community, district and national) to make effective, malaria control a priority.
5) At international level. raise additional funds for country activities, and focus support on 10 of the most severely-affected countries in the next two years.
6) Take lead responsibility for developing an impregnated bednet resource network.

UNDP will

1) Create catpacity for integration of malaria-related action into national poverty eradication policies, strategies and programmes.
2) Strengthen, through Sustainable Human Development activities. the balance of action among state, private sector, civil society and communities themselves, to ensure that people have access to basic social services anu productive assets.
3) Work through the UN Resident Coordinator system to encourage collaborative programming in support of intersectoral action and resource mobilization.

At regional/sub-regional levels, UND? will support links between Sub-regional Resource Facilities (SURFs), providing technical referral services to country offices and the Roll Back Malaria resource support works, and collaborate with WHO Regional Offices to strengthen capacity of relevant regional inter-governmental organizations (ISO) in support of Roll Back Malaria.

At global level, UNDP is providing continuing support for the UNDP/World Bank/WHO Special Programme for Research & Training in Tropical Diseases (TDR), which has as a major focus the development of drugs and tools for malaria control and adapting research in local settings.

The World Bank Group strongly supports the Roll Back Malaria global partnership. Malaria has a major impact on social and economic development. The Bank **has committed to**

1) Increasing World Bank investments in malaria control and research
2) Facilitating resource mobilization to support RBM
3) Enhancing a more effective involvement of Departments of Finance, Economics, Intfrastructure, Agriculture and others to become full partners in reducing malaria as a break on econumic growth
4) Exploring innovative finance mechanisms to deliver support
5) Supporting research on the economic aspects of malaria
6) Helping establish private-public partnerships with industry on new malaria products
7) Together with Roll Back Malaria partners, the Bank will actively pursue these activities through its country programmes and research agendas. Malaria must be reduced as a negative factor on macroeconomic growth.

RBM's first focus : Africa

The Roll Back Malaria campaign will focus first on Africa. It is aimed at:

1. Upgrading health delivery systems at both the local and national levels in malarious countries
2. Intensifying use of bed-nets (nets coated with insecticide) to prevent night-time biting by malaria-carrying mosquitoes
3. Mapping of malaria regions and of medical facilities to better direct health resources; developing new drugs for victims already infected with malaria
4. Coordinating the development and testing of new malaria drugs and vaccines; developing methods to address malaria in emergencies, (e.g. refugee and post-war situations).
5. At country level, RBM will work towards development of sustained capacity to address malaria (and other priority health problems) that is adapted to local realities, and delivering measurable and properly validated results.
6. RBM will support the building of coalitions for action at regional and country level, and assist with development of clear, evidence based

action plans at country and regional levels. RBM will develop a systematic approach to monitor progress and results, and broker financial and technical inputs into countries.

7. RBM will support Resource Networks which will facilitate the implementation of RBM in endemic countries by providing support in specialised areas, e.g.:

   a) Needs assessment and intervention at district level;
   b) Sector-wide approaches and financing;
   c) Quality and supply of anti-malarials at the local level;
   d) Implementation of bed net programmes, including supply of nets and insecticides;
   e) Improving quality of care at the home;
   f) Geographic mapping of malaria and health care;
   g) Prevention and control of epidemics;
   h) Monitoring of drug and insecticide resistance;
   i) Malaria control in war-torn zones.

RBM will create a network of teams to go into villages and analyze treatment and prevention practices at the household and community level, the availability and quality of health care by the public and private sector, and potential local partners.

RBM will provide technical and financial support for each analysis through this network at the district level.

In African districts with stable, high transmission malaria, RBM will simultaneously seek to significantly improve early diagnosis and appropriate treatment of malaria- related fevers in children, early treatment/prevention in pregnant women, and personal protection for children and pregnant mothers through the use of insecticide impregnated bed-nets (IIBNs). In many districts, this will require reinforcement of the local public and private health sector, focusing on activities at the community level. RBM will also attempt to upgrade the training of health care providers to enure quality care after the campaign ends.

RBM will set up a resource network throughout Africa to forecast malaria epidemics and their prevention. The resource network will also be used to track the quality and supply of drugs used to treat malaria.

Geoeraphic mapping of malaria and health care

For countries participating in RBM, national malaria information will be integrated with regional information to produce a comprehensive national malaria control map, as part of the international mapping of the disease. The information will allow a better estimation of the burden of malaria and the population at risk, and hence a better assessment for RBM. It will also provide more reliable and area-specific information for national and international advocacy for malaria control. Where RBM operations have started, information on the availability and quality of health services and the results of monitoring and evaluation will be added to the data base.

It is envisaged that till the end of 1999, RBM will have :

a) Supported countries in Africa to develop implementation plans for high transmission, stable malaria, that meet the overall objectives of RBM
b) Advanced plans for other malaria situations, i.e., epidemic malaria and malaria in other regions of the world.

The general objective of RBM will be to significantly reduce the global burden of malaria through interventions adapted to local needs and by reinforcement of the health sector. Goals are to be set by countries based pn situation analyses and assessment of feasibility, and could include :

a) Malaria morbidity and mortality goals
b) Financial goals (e.g., significant increase in resources available for community level activities in health care)
c) Accessibility goals (e.g., Percentage of population with access to early and adequate treatment)
d) Coverage goals (e.g. Proportion of the targeted population with insecticide treated bed nets)
e) Health sector reform goals (e.g. New partnerships with private sector health care providers)
f) Goals of policy change (e.g, Significant changes in policy favouring evidence - based strategy development).

6. WHO's New programme - "Tobacco Free Initiative"

Introduction

"Tobacco is a killer. Tobacco should not be advertised, subsidized or glamorized". These were the words of Dr. Gro Harlem Brundtland, WHO's new Director-General, while addressing the World Health Assembly at Geneva on 13th May 1998. At a meeting in the Woodrow Wilson Center in Washington on 22nd September 1998, she said, "Smoking is a communicated disease. The allure of smoking is communicated through advertising and peer pressure. The price is paid in lost heath and lost lives". The Director-General established a Cabinet Project, the Tobacco Free Initiative (TFI), in July 1998 to coordinate an improved global strategic response to tobacco as an important public health issue.

Why Focus on Tobacco?

1) **Public health impact**

   According to WHO estimates, there are currently 4 million deaths a. figure expected to rise to about 10 million by 2030. By that day smoking trends, tobacco is predicted to be the leading cause of world, causing about one in eight deaths. 70% of those deaths will occur in developing countries. The sheer scale of tobacco's impact on global particularly what is likely to happen without appropriate intervention in developing countries, is often not fully appreciated, Mortality data do not reflect additional toll caused by tobacco that is felt in terms of morbidity, disability and sufering among children and adults.

2) **Over a billion smokers**
   There are currently over a billion smokers in the world. The largest number is in Asia. Recent trends indicate that the smoking prevalence rate in adolescent boys and girls is rising in many countries.

3) **Tobacco use is bad economics**
   Large direct, indirect and intangible costs associated with tobacco hamper economic development rather than promote it.

4) **Tobacco harms the environment**
   In many of the tobacco growing countries evidence indicates negative environmental impacts of tobacco on agriculture, particularly when associated with deforestation required to increase farmland and cure tobacco plants.

Effective policies and interventions have been successful

Effective policies and interventions make a real difference to tobacco prevalence and consumption, and associated health outcomes. Most of the documented successes have occurred in developed countries where effective approaches have been implemented for several years. In more recent years, several developing countries have introduced similar measures. Early indications are that they too will be effective. The combined impact of legislation, increased tax and comprehensive community-based strategies on tobacco consumption in adults in Finland has led to significant reduction in tobacco use. Further, it should be noted that when Finland began addressing tobacco, it was not a wealthy country.

The lone-term mission of global tobacco control is to reduce smoking prevalence and tobacco consumption in all countries and among all groups, and thereby reduce the burden of disease caused by tobacco. In support of this mission, the goals of the Tobacco Free Initiative are:

1) To Galvanize global support for evidence-based tobacco control policies and actions.
2) Build new, and strengthen existing partnerships for action.
3) Heighten awareness of the social, human and economic harm of tobacco in all sectors of society, and the need to take comprehensive actions at all levels.
4) Accelerate national, regional and global strategic planning, implementation and evaluation.
5) Commission policy research to support rapid, sustained and innovative actions.
6) Mobilize adequate resources to support action.
7) Integrate tobacco into the broader agenda of health and development.
8) Facilitate the development of an effective Framework Convention for Tobacco Control and related protocols.

In achieving these goals, the Tobacco Free Initiative will build strong internal and external partnerships "with a purpose" with each WHO Cluster (WHO's administrative divisions have now been regrouped

into clusters) and Regional and Country Offices, and with a range of organizations and institutions around the world. The purpose of these partnerships will reflect the unique and complementary roles of WHO's partners and of WHO at all levels of the organization. Success will be measured in terms of actions achieved at local, country and global levels that lead to better tobacco control.

The Tobacco Free Initiative will take a global leadership role in promoting effective policies and interventions that make a real difference to tobacco prevalence and associated health outcome. Despite the seriousness of the problem, there is evidence to show that countries that, undertake concerted and comprehensive actions to address tobacco control can bring about significant reductions in tobacco related harm.

More resources for tobacco control activities

Since 22 July 1998, new resources have become available for international tobacco control. WHO has increased its financial commitment in its regular budgets, and a grant from the United Nations Foundation for a joint WHO/UNICEF project that focuses on tobacco use and its impact on children. A number of private foundations also have indicated potential areas of future support.

WHO's partners in the initiative

UNICEF : Supports programs to prevent children and adolescents fronl starting tobacco.

United United Nations Radio: Multilingual global communications.

World Bank : Effective use of excise tax and analyses of economic impacts.

CDC : Support for global surveillance and public service announcements.

EPA : Reaching environmental constituencies.

NIH (USA), IDRC (Canada), SIDA(Sweden) : Expanding the evidence-base through policy research.

International NGOs : Strengthening the grassroots; networking through the internet; media advocacy; communications.

Private sector : Energy and expertise from pharmaceuticals, media and entertainment industries.

Academic centers : Building capacity in several disciplines; research for action.

Partnerships within WHO

Cluster (New grouping of WHO's previous divisions)	Tobacco specific action
Evidence	Epidemiology of tobacco use
	Cost-effectiveness of interventions
Sustainable development	ETS/public places
	International conventions
Non-communicable Diseases	Colnmunity-based interventions
	Clinical management
	Operational research
Communicable diseases	TB control
Social change and Mental health	Primary prevention of substance abuse,
	Routes and roots of addiction
	Health promotion
	Healthy ageing
	Fire and injury prevention
Health systems and Community	Life-span approaches
	Child, adolescent and women's health
	Health professional's involvement
Health technology	Pharmaceutical aids to smoking cessation
Regulation of nicotine	UN, World Bank, NGOs, Private sector

7. Syndromic Approach

DEFINITION

Syndromic Approach is a method of managcment and control of diseases where the diagnosis and treatment is on the basis of groups of symptoms or signs, rather than for specific diseases. Treatment is targeted towards all diseases that could cause the signs and symptoms peculiar to the syndrome. Whereas, in the **etiologic approach** the patient is diagnosed and treated for the specific disease. This approach needs the services of specialists and requires laboratory support. Sexually transmitted diseases can be easily managed at the primary health care level by the syndromic approach e.g. the health worker will provide :

1. Combined treatment for **genital ulcer,** rather than specifically diagnose and treat syphilis or chancroid.
2. Combined treatment for **urethral discharge,** rather than specifically diagnose and treat gonorrhea in men.
3. Combined treatment for **vaginal discharge,** rather than specifically diagnose and treat gonorrhea, candidiasis, trihomonas or gardnarella in women.
4. Combined treatment for **Pelvic inflammatory disease (PID)** in women with lower abdominal pain, rather than diagnose and trcat the specific cause of PID.

The patient's sexual history and the knowledge about the endemicity of certain diseases in the area are other factors, which are of help in the syndromic approach.

STANDARD CASE DEFINITIONS Standard Case Definitions have to be developed for the syndromic approach to make it easy for the primary health care workers to make a correct syndromic diagnosis e.g.

Genital Ulcer: The presence of an ulcer(s) in the genital region with or without associated lympadenopathy.

Urethral Discharge : The presence of discharge from the urethra of a male patient, which may be spontaneous or appear an milking the urethra.

Vaginal Discharge : The presence of vaginal discharge, which is abnormal in colour, odour and amount, which is visible with or without speculum examination.

Pelvic Inflammatory Disease (PID): The complaint of lower abdominal pain in a women which is accompanied by the presence of abnormal discharge, body temperature more than 38°C and pain on moving the cervix.

ADVANTAGES OF SYNDROMIC APPROACH

1. It is relatively simple to make a syndromic diagnosis and primary health care workers can make a syndromic diagnosis as well as treat the patient on the basis of standard treatment regimen, without having to wait for laboratory tests as in the case of etiologic diagnoses.
2. This approach helps in reducing the waiting period, the number of visits by the patient to the health centre, as well as, the social stigma, which may be associated with certain diseases like the sexually transmitted diseases. Reduction of waiting time also helps in rendering the patient non-infectious much earlier.
3. It is a very cost-effective method for the developing countries where the laboratory support for etiologic diagnosis is available only at secondary and tertiary level centres.

DISADVANTAGES

1. Tendency to over treat, resulting in wastage of already scarce drugs.
2. There are chances of the development of drug resistance due to the possibility of indiscriminate use of drugs.
3. Many patients are often asymptomatic and hence will escape treatment in syndromic approach.
4. Follow up of patients is usually difficult in this approach since the patient is treated in a single visit.
5. Although it is very convenient for countries with a shortage of trained health care personnel and laboratory facilities many specialist physicians do not accept it as a scientific method.
6. Some senior health administrators also, look upon this approach as ethically unacceptable since it empowers relatively untrained health care personnel to treat patients at their own discretion.

8. New challenges to Public Health : Emerging Infection

Public Health has seen some exciting challenges, in the last couple of years. The global village is slowly awakening to the realities of globalisation. Newly emerging infectious diseases like SARS, BSE, Leptospirosis and Bird Flu have sent even the most developed countries into a tizzy. This chapter, which is an addition to the reprint, will take a brief look at the important public health aspects of these diseases. The WHO also recently adopted the 'Framework Convention for Tobacco Control (FCTC)'. This is a landmark convention since WHO has for the first time used its authority to enact an international public health legislation for ensuring the health protection of member countries. Students should expect these questions in both their practical and theory exams.

SARS

Introduction: Severe Acute Respiratory Syndrome (SARS) affected a large number of countries in 2003. WHO has issued a global alert for SARS. The viral infection is a newly defined clinical illness, which is characterized by atypical pneumonia. Available evidence suggests that SARS emerged in the Guangdong Province, in Southern China. More than one third of the early cases were in food handlers.

Countries that reported SARS Cases: A cumulative total of 8422 probable cases, with 916 deaths were reported from 29 countries in 2003. Of these, 5327 cases and 349 deaths were reported from China. Other countries that suffered the major brunt were Singapore, Taiwan, Viet Nam and Canada. WHO announced that the last chain of human transmission was broken on 5 July 2003.

Disease agent: Caused by a type of coronavirus termed SARS-CoV.

Transmission: Symptomatic cases are highly infectious. The virus appears to spread through close contact as has been shown by the occurrence of a large number of cases in health care workers and immediate family members. The primary mode of transmission appears to be direct mucous membrane (eyes, nose and mouth) contact with infectious respiratory droplets and /or through exposure to fomites. Transmission efficiency appears to be greatest from severely ill patients or those experiencing rapid clinical deterioration. Data from Singapore shows that very few secondary cases occur when symptomatic cases are isolated within 5 days of onset of illness. Virus excretion from the respiratory tract peaks at around day 10 of the illness and then declines. Virus shedding in stools begins later than in respiratory secretions but also follows an inverted 'V' distribution (peaks around days 12-14 and then declines. Procedures that promote aerosolization of infectious respiratory droplets or other potentially infectious materials (such as feces and urine) in hospitals or other settings may amplify transmission. There is no evidence of feco-oral transmission at this stage. Several animal coronaviruses are transmitted by the feco-oral route and hence this route has to be kept in mind. A significant observation is that children are rarely affected. Further investigations are required to determine whether children have asymptomatic or mild infections. There has also been limited transmission associated with air travel that forced WHO to issue an emergency travel advisory to airlines and travelers.

The slaughter of wildlife for human consumption in the wet markets of southern China is associated with serological evidence of infection (There is evidence that natural infection with SARS-CoV in a number of animal species indigenous to China and parts of south-east Asia. Studies have detected several closely genetically related coronaviruses in wild animals like palm civet, raccoon dog and Chinese ferret badger. These and other wild animals are considered and sold as traditional delicacies in the markets of southern China). The transmission of SARS in the Metropole Hotel and Amoy Gardens in Hong Kong has been attributed to environmental contamination and also a possible animal vector (a number of pet and dogs and cats living in the complex tested positive for SARS-CoV).

The virus is stable in faeces and urine at room temperature for 1-2 days. It is stable for up to 4 days in stool from patients with diarrhoea because of its higher PH compared to normal stool. The virus loses infectivity after exposure to different commonly used disinfectants and fixatives. Heat at 56° C rapidly kills 10,000 units of SARS-CoV per 15 minutes.

Incubation Period: Range 1-14 days, Median 4-5 days and Mean 4-6 days. Whether the route of transmission influences the incubation is still unclear.

Case Fatality : SARS is associated with substantial morbidity and mortality. The crude CFR is around 15% (range 0-50%, depending on the age group (older), sex (male), presence of co-morbidity, health seeking behavior and the level of care available). A global case fatality ratio of 11% was recorded at the end of the outbreak.

SARS Case Definitions (issued by WHO on 1 May 2003)

Suspect Case

1. A person presenting after 1 November 2002 with history of :
   a) High fever (>38 °C).

   AND
   b) Cough or breathing difficulty.
   AND one or more of the following exposures during the 10 days prior to onset of symptoms:
   c) Close contact with a person who is a suspect or probable case of SARS;
   d) History of travel, to an area with recent local transmission of SARS.
   e) Residing in an area with recent local transmission of SARS.

   (1 November 2002 is considered as the starting point for SARS surveillance. International transmission of SARS was first reported in March 2003 for cases with onset in February 2003. 'Close Contact' in the context of SARS is defined as a person who has cared for, lived with, or had direct contact with respiratory secretions or body fluids of a suspect or probable case of SARS)
2. A person with an unexplained acute respiratory illness resulting in death after 1 November 2002, but on whom no autopsy has been performed
   AND one or more of the following exposures during to 10 days prior to onset of symptoms:
   a) Close contact, with a person who is a suspect or probable case of SARS.
   b) History of travel to an area with recent local transmission of SARS.
   c) Residing in an area with recent local transmission of SARS.

Probable Case

1. A suspect case with radiographic evidence o infiltrates consistent with pneumonia o respiratory distress syndrome (RDS) on chest X-ray (CXR).
2. A suspect case of SARS that is positive for SARS coronavirus by one or more assays.
3. A suspect case with autopsy findings consistent with the pathology of RDS without an identifiable cause.

Exclusion criteria: A case should be excluded if an alternative diagnosis can fully explain their illness.

Reclassification of cases: As SARS is currently a diagnosis of exclusion; the status of a reported case may change over time. A patient should always be managed as clinically appropriate, regardless of their case status.

a) A case initially classified as suspect or probable, for which an alternative diagnosis can fully explain the illness, should be discarded after carefully considering the possibility of co-infection.
b) A suspect case that, after investigation, fulfils the probable case definition should be reclassified as "probable".
c) A suspect case with a normal CXR should be treated, as deemed appropriate, and monitored for 7 days. Those cases in which recovery is inadequate should be re-evaluated by CXR.
d) Those suspect cases in which recovery is adequate but whose illness cannot be fully explained by an alternative diagnosis should remain as "suspect".
e) A suspect case who dies, on whom no autopsy is conducted, should remain classified as "suspect". However, if this case is identified as being part of a chain transmission of SARS, the case should be reclassified as "probable".
f) If an autopsy is conducted and no pathological evidence of RDS is found, the case should be "discarded".

Management of Severe Acute Respiratory Syndrome (SARS)

Management of Suspect and Probable SARS Cases

- Hospitalize under isolation or cohort with other suspect or probable SARS cases
- Take samples (sputum, blood, sera, urine,) to exclude standard causes of pneumonia

(including atypical causes); consider possibility of co-infection with SARS and take appropriate chest radiographs.

- Take samples to aid clinical diagnosis SARS including:

  White blood cell count, platelet count, creatine phosphokinase, liver function tests, urea and electrolytes, C reactive protein and paired sera. (Paired sera will be invaluable in the understanding of SARS even if the patient is later not considered a SARS case)
- At the time of admission the use of antibiotics for the treatment of community-acquired pneumonia with atypical cover is recommended.
- Pay particular attention to therapies/ interventions, which may cause aerosolization such as the use of nebulisers with a bronchodilator, chest physiotherapy, bronchoscopy,gastroscopy, any procedure/intervention, which may disrupt the respiratory tract. Take the appropriate precautions (isolation facility, gloves, goggles, mask, gown, etc.) if you feel that patients require the intervention/ therapy.
- In SARS, numerous antibiotic therapies have been tried with no clear effect. Ribavirin with or without use of steroids has been used in an increasing number of patients. But, in the absence of clinical indicators, its effectiveness has not been proven. It has been proposed that a coordinated multicentred approach to establishing the effectiveness of ribavirin therapy and other proposed interventions be examined.

Management of Contacts of Probable SARS Cases

- Give information on clinical picture, transmission, etc. of SARS to the contact
- Place under active surveillance for 10 days and recommend voluntary home isolation
- Ensure contact is visited or telephoned daily by a member of the public health care team
- Record temperature daily
- If the contact develops disease symptoms, the contact should be investigated locally at an appropriate health care facility
- The most consistent first symptom that is likely to appear is fever

Management of Contacts of Suspect SARS Cases : As a minimum the following follow up is recommended:

- Give information on clinical picture, transmission etc of SARS to the contact
- Place under passive surveillance for 10 days
- If the contact develops any symptoms, the contact should self report via the telephone to the public health authority
- Contact is free to continue with usual activities
- The most consistent first symptom which is likely to appear is fever

Removal of 'Contact' from Follow up: If as a result of investigations, suspected or probable cases of SARS are discarded (no longer meet suspect or probable case definitions) then contacts can be discharged from follow up.

SARS Prevention: Transmission can be dramatically reduced with:

1. Public information and education to encourage prompt reporting of symptoms.
2. Case identification, prompt isolation and proper barrier nursing techniques.
3. Vigorous contact tracing and voluntary home quarantine of close contacts for the duration of the incubation period.

Variant Creutzfeldt-Jakob Disease

Introduction: Variant CJD (vCJD) is a rare, degenerative, fatal brain disorder in humans. Since 1996, strong evidence has accumulated for a causal relationship between ongoing outbreaks in Europe of disease in cattle called Bovine Spongiform Encephalopathy (BSE, or "mad cow disease") and a disease in humans called variant CJD (vCJD). Both disorders are invariably fatal brain diseases with unusually long incubation periods measured in years and are caused by an unconventional mode of transmission. Although there is very strong evidence that the agent responsible for the human disease is the same agent responsible for the BSE outbreaks in cattle, the specific foods that might be associated with the transmission of this agent from cattle to humans are unknown.

Countries reporting vCJD : As of December 1, 2003, a total of 153 cases of vCJD had been reported in the world: 143 from the United Kingdom, six from France, and one each from Canada, Ireland, Italy, and the United States (note: the U.S. case was reported in a patient who lived in the United Kingdom before moving to the United States). Almost all the 153 patients had multiple-year exposures in the United Kingdom between 1980 and 1996 during the occurrence of a large UK outbreak of Bovine Spongiform Encephalopathy (BSE, commonly known as mad cow disease) among cattle.

Transmission: It is believed that the persons who have developed vCJD became infected through their consumption of cattle products contaminated with the agent of BSE. There is strong evidence and general agreement that the cattle infection may have resulted from the feeding infected rendered bovine meat-and-bone meal to cattle. There has never been a case of vCJD that did not have a history of exposure within a country where this cattle disease, BSE, was occurring. Evidence till date indicates that there has never been a case of vCJD transmitted through direct contact of one person with another. However, a case of probable transmission of vCJD through transfusion of blood components from an asymptomatic donor who subsequently developed the disease has been reported.

Incubation period: The incubation period for vCJD is unknown because it is a new disease. However, it is likely to be many years or decades from the point of infection.

Clinical Features: vCJD Differs from Classic CJD. This variant form of CJD should not be confused with the classic form of CJD that is endemic throughout the world.

1. Predominantly affects younger people. The median age at death of patients with classic CJD is 68 years, and very few cases occur in persons under 30 years of age. In contrast, the median age at death of patients with vCJD is 28 years.
2. Prominent psychiatric or sensory symptoms at the time of clinical presentation and delayed onset of neurological abnormalities, including ataxia within weeks or months, dementia and myoclonus late in the illness, a duration of illness of at least 6 months, and a diffusely abnormal non-diagnostic electro-cephalogram.

Diagnosis: Diagnosis is on the basis of the typica signs and symptoms and progression of th disease. In most CJD patients, the presence of 14 3-3 protein in the cerebrospinal fluid and/or a typical electroencephalogram (EEG) pattern, both of which are believed to be diagnostic for CJD, have been reported. However, a confirmatory diagnosis of CJD requires neuropathologic and/or immunoassay of brain tissue obtained either at biopsy or autopsy.

Treatment: There is no known treatment of vCJD and it is invariably fatal.

Prevention: Public health control measures, such as enhanced surveillance, the culling of sick animals, and bans of specified risk materials (SRM), have been instituted in European countries to prevent potentially infected meat and tissues from entering the human food chain. The most stringent of these control measures, including an "Over Thirty Months Scheme" that excludes all animals older than 30 months from the human food and animal feed chains, have been applied in the United Kingdom and appear to be highly effective. In June 2000, the European Union Commission on Food Safety and Animal Welfare strengthened the European Union's set of control measures in relation to BSE by adopting a decision requiring all member states to remove SRMs from the animal feed and human food chains as of October 1, 2000; such bans had already been instituted in most member states. Also noteworthy among European Union's set of control measures are the banning of the use of mechanically recovered meat from the vertebral column of cattle, sheep, and goats for human food, and the BSE testing of all cattle aged over 30 months destined for human consumption.

To reduce the possible current risk of acquiring vCJD from food, travelers to Europe or other areas with indigenous cases of BSE or at possibly increased risk of BSE should consider:

1. Avoiding beef and beef products altogether.
2. Selecting beef or beef products, such as solid pieces of muscle meat (versus calf brains or beef products such as burgers and sausages), which might have a reduced opportunity for contamination with tissues that may harbor the BSE agent.
3. Milk and milk products from cows are not believed to pose any risk for transmitting the BSE agent.

Leptospirosis

Introduction: Leptospirosis is the most wide spread Zoonosis in the world. Apart from humans, Leptospirosis affects domestic animals (dogs, cattle, pigs and live stock) in whom the infection is mild. It is found in rodents, mongoose and several wild animals. Leptospirosis is also known as Cane-Cutter Fever, Canicola Fever, Hemorrhagic Jaundice, Icterohemorrhagic Fever, Mud Fever, Rice-Field Fever, Stuttgart Disease, Swamp Fever, Swineherd's Disease and Weil Disease

Transmission: Coming in contact with water, soil or mud and food that has been contaminated with the infected animal's urine is responsible for infection in Man. Stagnant water as well as rapidly flowing water can be the source of infection. Cases increase after heavy rains and flooding. Leptospirosis is an occupational hazard to sewage workers, abattoir workers and farm hands. Transplacental spread is common in rodents and farm animals and may result in abortions. Transplacental transmission resulting in abortions is also reported in humans. In humans the organism gains entry through wounds in the skin also through percutaneous infection. Certain serotypes are transmitted from mother to infants via breast milk (S. Hardjo). Transmission by ingestion is uncommon in humans but certain serotypes have caused epidemics through contaminated drinking water. Insects passively transmit the organism on to food / water (or) by being rubbed into the skin of the humans. Infections have been reported when animals (dog, rat or ferrets) have bitten humans.

Reservoirs: In the epidemiology of Leptospirosis the chronic persistent carriers and shedders of virulent leptospirae are important. A biological equilibrium exists between some strains and their hosts. (e.g. Icterohaemorrhagiae and rats).

Disease Agent: The genus Leptospirae consists of 2 complexes, one of which L. Interrogans complex is pathogenic to humans and animals while L. Biflexa is non pathogenic. Sero-groups responsible for human disease include:

L. Ictohaemorrhagiae
L. Canicola
L. Autumnalis
L. Australis
L. Andamana
L. Patoc

Survival: Pathogenic Leptospirae survive outside the animal body for 21 days or longer in a moist, warm and neutral PH environment. Leptospirae are killed by increase in salinity, excessive sunlight and dry weather.
Incubation period of 2 to 26 days (average 10 days)

Clinical features:
Abrupt onset of fever, rigors, myalgias, and headache in 75 to 100% of patients, dry cough (25-35% of cases), nausea, vomiting, and diarrhea (50% of cases). Less common symptoms include joint aches, bone pain, sore throat, and abdominal pain, conjunctivitis. Approximately 7 to 40% of patients may have muscle tenderness, an enlarged spleen or liver, enlarged lymph glands, sore throat, muscle rigidity, abnormal lung sounds, or skin rash.

Laboratory Diagnosis:

1. White blood cell (WBC) counts are generally less than 10,000.
2. Urin analysis is frequently abnormal.
3. Elevated creatine kinase is found in approximately 50% of patients.
4. About 40% of patients have minimal to moderate elevations of liver enzymes.
5. Diagnosis is most frequently made by serologic (antibody) testing.
6. Leptospira are best visualized by dark field microscopy, silver stain, or fluorescent microscopy.
7. Unlike *Treponema pallidum*, *Leptospira* can be grown from blood, urine, and CSF. It is slow growing and the laboratory needs to be notified.
8. Isolation of the organism from the blood is successful in 50% of cases.
9. Urine cultures become positive during the second week of the illness and remain so for up to 30 days.

Treatment: Depending on sensitivity Leptospirosis can be treated with Penicillins, Tetracyclines, Chloramphenicol, and Erythromycin. Supportive care may be required in complicated cases.

Prevention: Avoid possibly infected water and where it is unavoidable Doxycycline can be used for Chemoprevention can be done with.

Bird Flu-Avian Influenza Outbreak (2003-04)

Introduction: An outbreak of avian influenza more commonly known as bird flu, is affecting bird populations in countries throughout Asia. The outbreak is caused by the H5N1 subtype of influenza A. Human cases have also been reported.

Countries reporting outbreaks :

1. **In birds**: Outbreaks of avian influenza A (H5N1) have been confirmed among poultry in Cambodia, China, Hong Kong (in a single peregrine falcon), Indonesia, Japan, Laos, South Korea, Thailand, and Vietnam.
2. **In people**: The outbreak of bird flu has resulted in human cases of H5N1 infection in Vietnam and Thailand. Deaths have been reported. At this time it is believed that these cases resulted from contact with infected birds or surfaces contaminated with excretions from infected birds. An investigation is ongoing to determine the source of human infections.

Disease Agent: Influenza A (H5N1) is a subtype of the Type A influenza virus. Wild birds are the natural hosts of the virus, hence the name avian influenza or bird flu. The virus was first isolated from birds (terns) in South Africa in 1961. The virus circulates among birds worldwide. It is very contagious among birds and can be deadly to birds, particularly domesticated birds like chickens.

The H5N1 strain implicated in the current outbreak has been genetically sequenced. Following is a summary of what has been learned:

1. All genes are of bird origin. This means that the virus has not acquired genes from human influenza viruses, a development that would make person-to-person spread more likely.
2. There are likely different variations of H5N1 virus circulating at this time. Genetic sequencing of virus samples from South Korea and Vietnam show that the viruses in these two countries are slightly different.
3. Genetic sequencing of A (H5N1) virus samples from human cases in Vietnam and Thailand show antiviral resistance to amantadine and rimantadine, two of the antiviral drugs commonly used for influenza. The remaining two antivirals (oseltamavir and zanamavir) should still be effective against this strain of H5N1.

(There are 3 types of influenza viruses: A, B and C. Influenza type A viruses are divided into subtypes based on two proteins on the surface of the virus. These proteins are called hemagglutinin (HA) and neuraminidase (NA). There are 15 different HA subtypes and 9 different NA subtypes. The viruses are named according to their surface proteins. For example, an "H7N2 virus" designates an influenza A subtype that has a hemagglutinin 7 protein and a neuraminidase 2 protein. Wild birds are the natural host to all subtypes of influenza A viruses. Influenza B and certain subtypes of influenza A (H1N1, H1N2 and H3N2) normally circulate among humans and cause yearly epidemics of disease. Type C influenza viruses are milder and do not cause epidemics. Type A viruses historically have been the ones responsible for influenza pandemics. In addition to birds, type A influenza viruses can infect several animal species, including pigs, horses, seals and whales. Types B and C influenza viruses do not affect domestic animals).

Transmission: The virus does not typically infect humans. In 1997, however, the first instance of direct bird-to-human transmission of H5N1 was documented during an outbreak of avian influenza among poultry in Hong Kong; the virus caused severe respiratory illness in 18 people, of whom 6 died. Since that time, there have been other instances of H5N1 infection among humans. H5N1 viruses have not been capable of efficient human-to-human transmission so far. This is something that is being watched carefully and is being investigated during this outbreak.

Infected birds shed virus in saliva, nasal secretions and feces. Avian influenza viruses spread among susceptible birds when they have contact with contaminated excretions. It is believed that most cases of H5N1 infection in humans have resulted from contact with infected poultry or contaminated surfaces.

Clinical features: Published information about the clinical course of human infection with H5N1 avian influenza is limited to studies of cases in the 1997 Hong Kong outbreak. In that outbreak, patients developed typical influenza-like symptoms (e.g., fever, cough, sore throat and muscle aches) to eye infections, pneumonia, acute respiratory distress, viral pneumonia, and other severe and life-threatening complications. In several of the fatal cases, severe respiratory distress secondary to viral pneumonia was seen.

Previously healthy adults and children, and some with chronic medical conditions, were affected.

Health-care providers should be alert for respiratory illness among persons who may have been exposed to infected poultry. The following section provides recommendations for health-care professionals who may need to evaluate symptomatic persons with possible avian influenza exposure.

- Persons who develop a febrile respiratory illness should have a respiratory sample (e.g., nasopharyngeal swab or aspirate) collected.
- The respiratory sample should be tested by RT-PCR for influenza A, and if possible for H1 and H3. If such capacity is not available in the state, or if the result of local testing is positive, then CDC should be contacted and the specimen should be sent to CDC for testing.
- Virus isolation should not be attempted unless a biosafety level 3+ facility is available to receive and culture specimens.
- Optimally, an acute- (within 1 week of illness onset) and convalescent-phase (after 3 weeks of illness onset) serum sample should be collected and stored locally in case testing for antibody to the avian influenza virus should be needed.
- Requests for testing should come through the state and local health departments, which should contact the CDC Director's Emergency Operations Center at 770-488-7100 before sending specimens for testing.

Treatment: Antiviral drugs, some of which can be used for both treatment and prevention, are clinically effective against influenza A virus strains in otherwise healthy adults and children, but have some limitations. Some of these drugs are also expensive and supplies are limited.

Prevention: Key to containing the outbreak is the culling (killing) of sick and exposed birds. This was done to contain the 1997 H5N1 outbreak in Hong Kong. Many experts believe this was crucial to averting many more human cases. For the current outbreak in Asia, governments are culling poultry to try to contain the virus. Patients are being treated and isolated, and investigations are underway to determine the source of infection. Travelers to countries in Asia with documented H5N1 outbreaks should avoid poultry farms, contact with animals in live food markets, and any surfaces that appear to be contaminated with feces from poultry or other animals. Most influenza experts agree that the prompt culling of Hong Kong's entire poultry population in 1997 probably averted a pandemic.

Precautions for Individuals Participating in Avian Influenza Outbreak Control and Eradication Activities: Persons involved in outbreak control and eradication activities (e.g., euthanasia, carcass disposal, and cleaning and disinfection of premises affected by avian influenza) on poultry farms or live bird markets are at increased risk for exposure to avian influenza. Such persons often have prolonged, direct contact with infected birds and/or contaminated surfaces in an enclosed setting. Interim guidance to reduce the risk of transmission, include personal protective equipment, vaccination with seasonal influenza vaccine, administration of antiviral drugs for prophylaxis, surveillance and monitoring of workers, and evaluation of workers who develop a febrile respiratory illness within 7 days of their last exposure.

Precautions for Other Individuals with Possible Exposure to Avian Influenza: The risks for exposure to avian influenza viruses and the possibility of viral reassortment would be expected to be lower for persons with more routine (i.e., less intense and prolonged) occupational or other types of contact with poultry or contaminated surfaces or equipment on affected farms or in live bird markets. Individuals who develop a febrile respiratory illness within a week after their last exposure to avian-infected or exposed birds or potentially contaminated surfaces should consult a health-care provider. Before visiting a health-care setting, tell the health care provider about symptoms and recent possible exposures to avian influenza.

Food Safety: There is no evidence that any human cases of avian influenza have been acquired by eating poultry products. Influenza viruses such as H5N2, H7N2, and H5N1 are destroyed by adequate heat, as are other food borne pathogens. Consumers are reminded to follow proper food preparation and handling practices, including:

- Cook all poultry and poultry products (including eggs) thoroughly before eating. (This means that chicken should be cooked until it reaches a temperature of 180 degrees Fahrenheit, throughout each piece of chicken.)

- Raw poultry always should be handled hygienically because it can be associated with many infections, including salmonella. Therefore, all utensils and surfaces (including hands) that come in contact with raw poultry should be cleaned carefully with water and soap immediately afterwards.

Vaccine: Experience in the production of influenza vaccines is also considerable, particularly as vaccine composition changes each year to match changes in circulating virus due to antigenic drift. However, at least four months would be needed to produce a new vaccine, in significant quantities, capable of conferring protection against a new virus subtype.

Cause of Concern: All influenza viruses can change. It is possible that an avian influenza virus could change so that it could infect humans and could spread easily from person to person. Because these viruses commonly do not infect humans, there is little or no immune protection against them in the human population. If an avian influenza virus were able to infect people and gain the ability to spread easily from person to person then an "influenza pandemic" could begin. Based on historical patterns, influenza pandemics can be expected to occur, on average, three to four times each century when new virus subtypes emerge and are readily transmitted from person to person. However, the occurrence of influenza pandemics is unpredictable. In the 20th century, the great influenza pandemic of 1918–1919, which caused an estimated 40 to 50 million deaths worldwide, was followed by pandemics in 1957–1958 and 1968–1969. Experts agree that another influenza pandemic is inevitable and possibly imminent. An influenza pandemic is a global outbreak of influenza and occurs when a new influenza virus emerges, spreads, and causes disease worldwide. Once a new pandemic influenza virus emerges and spreads, it typically becomes established among people and circulates for many years. Past influenza pandemics have led to high levels of illness, death, social disruption and economic loss.

There were 3 pandemics in the 20th century. All of them spread worldwide within 1 year of being detected. They are:

1. **1918-19, "Spanish flu,"** [A (H1N1)], caused the highest number of known flu deaths: more than 500,000 people died in the United States, and 20 million to 50 million people may have died worldwide. Many people died within the first few days after infection and others died of complications soon after. Nearly half of those who died were young, healthy adults.
2. **1957-58, "Asian flu,"** [A (H2N2)], caused about 70,000 deaths in the United States. First identified in China in late February 1957, the Asian flu spread to the United States by June 1957.
3. **1968-69, "Hong Kong flu,"** [A (H3N2)], caused approximately 34,000 deaths in the United States. This virus was first detected in Hong Kong in early 1968 and spread to the United States later that year. Type A (H3N2) viruses still circulate today.

Framework Convention for Tobacco Control

The Framework Convention on Tobacco Control (FCTC) is the first treaty initiated by the World Health Assembly, the governing body of the World Health Organization (WHO). Negotiations began in October 1999 and concluded on March 1st 2003. The FCTC was adopted at the World Health Assembly on May 23, 2003 and is now open for signature and ratification. The objective of the FCTC is "to protect present and future generations from the devastating health, social, environmental and economic consequences of tobacco consumption and exposure to tobacco smoke." The FCTC provides Parties with a considerable degree of flexibility in implementing measures beyond those described in the treaty. Significantly, Article 2.1 of the FCTC states that all Parties are encouraged to implement measures that are stronger than the minimum standards required by the treaty.

Significant provisions of FCTC:

1. **Advertising, Promotion and Sponsorship (Article 13):** A comprehensive ban is required: The FCTC requires all Parties to undertake a comprehensive ban on tobacco advertising, promotion and sponsorship within five years of ratifying the treaty. The ban must include cross-border advertising originating within a Party's territory. The definitions of advertising, promotion and sponsorship are broad and include indirect as well as direct forms. Countries with constitutional constraints are required to restrict advertising, promotion and sponsorship, including cross-border advertising, in a manner consistent with their constitutional principles. The Parties also

agree to consider a protocol to elaborate on the cross-border provisions, for example the technical and legal aspects of preventing or blocking advertising on the internet and satellite television.

2. **Packaging and Labeling (Article 11):** Large health warning labels are required. Parties to the treaty agree that health warning labels ideally should cover 50% or more of the principle display areas of each packet, which for a standard cigarette package means both the front and back. Parties are required to implement health-warning labels that cover, at a minimum, 30% of the principle display areas within three years of ratifying the treaty. Health warning labels must include rotating messages in the principle languages of the Party, and may include pictures or pictograms. Deceptive labels must be prohibited. Countries agree to prohibit misleading or deceptive terms on tobacco product packages within three years of becoming a Party. Research has proved that cigarettes that are labeled "light", "low tar", and "mild" (among other terms) are as dangerous as those denoted as regular and thus these terms mislead and deceive consumers about the risks involved in the use of these products. Although the treaty does not specify the terms that Parties should ban, the scientific evidence would certainly support banning the use of terms such as "light", "mild", "low tar", etc.

3. **Secondhand Smoke (Article 8):** Nonsmokers must be protected in workplaces, public transport and indoor public places. The treaty recognizes that exposure to tobacco smoke has been scientifically proven to cause death, disease and disability. It requires all Parties to implement effective measures to protect nonsmokers from tobacco smoke in public places, including workplaces, public transport and indoor public places — evidence indicates that only a total smoking ban is effective in protecting non-smokers.

4. **Smuggling (Article 15):** Action is required to eliminate tobacco smuggling. Measures required include marking all tobacco packages in a way that signifies the origin and final destination or the legal status of the product, and cooperating with one-another in anti-smuggling, law enforcement and litigation efforts.

5. **Taxation & Duty Free Sales (Article 6):** Tobacco tax increases are encouraged. The treaty states that "each Party should take account of its national health objectives concerning tobacco control" in its tobacco tax and price policies. The treaty recognizes that raising prices through tax increases and other means "is an effective and important means of reducing tobacco consumption by various segments of the population, in particular young persons." Duty-free sales are discouraged. Parties may prohibit or restrict duty-free sales of tobacco products.

6. **Product Regulation & Ingredient Disclosure (Articles 9 & 10):** Tobacco products are to be regulated. The Parties agree to establish guidelines that all nations may use in regulating the content of tobacco products. Ingredients are to be disclosed. Parties shall require manufacturers to disclose to the government the contents of their tobacco products.

7. **Liability (Articles 4.5 and 19):** Legal action is encouraged as a tobacco control strategy. The treaty recognizes that liability issues are an important part of a comprehensive tobacco control program and the Parties agree to consider legislative and litigation approaches to advance tobacco control objectives. Parties also agree to cooperate with one another in tobacco-related legal proceedings.

8. **Treaty Oversight (Article 23):** A strong Conference of the Parties will oversee the treaty. The FCTC establishes a Conference of the Parties (COP), which will convene within one year of the treaty's entry into force. The COP is empowered to monitor the implementation of the treaty, adopt protocols, annexes and amendments to the FCTC, and to create appropriate subsidiary bodies to carry out specialized tasks.

9. **Financing (Article 26):** Parties have committed themselves to promote funding for global tobacco control. The Parties agree to mobilize financial assistance from all available sources for tobacco control initiatives for developing country Parties and for Parties with economies in transition, including from regional and international intergovernmental organizations and other public and private sources.

Other Important Commitments:

1. Each Party shall establish or reinforce and finance a national coordinating mechanism or focal point for tobacco control (Article 5).
2. Parties shall endeavor to include tobacco cessation services in their national health programmes (Article 14).
3. Parties shall prohibit or promote the prohibition of the distribution of free tobacco products(Article 16).
4. Parties shall promote the participation o NGOs in the development of national tobacc control programmes(Article 12).
5. Parties shall prohibit the sale of tobacc products to persons under the age set b national law, or eighteen(Article 16).
6. No reservations to the FCTC are allowe (Article 30).
7. The FCTC will enter into force 90 days afte ratification by the 40th country(Article 36).

9. Accredited Social Health Activitists (ASHA)

BACKGROUND: Under the National Rural Health Mission (NRHM) a new cadre of community based functionaries, named as Accredited Social Health Activist (ASHA) have been proposed in addition to the exisiting ANM and Anganwadi Workers. ASHA will be the first health contact for any health related demands of deprived sections of the population, especially women and children, who find it difficult to access health services.

ROLES & RESPONSIBILITIES:

1. ASHA will be a health activist in the community who will create awareness on health and its social determinants such as nutrition, basic sanitation & hygienic practices, healthy living and working conditions, information on existing health services and the need for timely utilization of health & family welfare services and mobilize the community towards local health planning and increased utilization and accountability of the existing health services to promote good health practices.
2. ASHA will counsel women on birth preparedness, importance of safe delivery, breast-feeding and complementary feeding, immunization, contraception and prevention of common infections including Reproductive Tract Infection/Sexually Transmitted Infection (RTIs/STIs) and care of the young child.
3. ASHA will also provide a minimum package of curative care as appropriate and feasible for that level and make timely referrals.
4. ASHA will mobilize the community and facilitate them in accessing health and health related services available at the village/sub-center/ primary health centers, such as Immunization, Ante Natal Check-up (ANC), Post Natal Check-up (PNC), ICDS, sanitation and other services being provided by the government.
5. She will work with the Village Health & Sanitation Committee of the Gram Panchayat to develop a comprehensive village health plan.
6. She will arrange escort/accompany pregnant women & children requiring treatment/ admission to the nearest pre-identified health facility i.e. Primary Health Centre/ Community Health Centre/ First Referral Unit (PHC/CHC / FRU).
7. ASHA will provide primary medical care for minor ailments such as diarrhea, fevers, and first aid for minor injuries.
8. She will be a provider of Directly Observed Treatment Short-course (DOTS) under Revised National Tuberculosis Control Programme.
9. She will also act as a depot holder for essential provisions being made available to every habitation like Oral Rehydration Therapy (ORS), Iron Folic Acid Tablet (IFA), chloroquine, Disposable Delivery Kits (DDK), Oral Pills & Condoms, etc. A Drug Kit will be provided to each ASHA. Contents of the kit will be based on the recommendations of the expert/technical advisory group set up by the Government of India.
10. Her role as a provider can be enhanced subsequently. States can explore the possibility of graded training to her for providing newborn care and management of a range of common ailments particularly childhood illnesses.
11. She will inform about the births and deaths in her village and any unusual health problems/ disease outbreaks in the community to the Sub-Centres/Primary Health Centre.
12. She will promote construction of household toilets under Total Sanitation Campaign.
13. Fulfillment of all these roles by ASHA is envisaged through continuous training and up-gradation of her skills, spread over two years or more.

SELECTION

1. The general norm will be 'One ASHA per 1000 population'. In tribal, hilly, desert areas the norm could be relaxed to one ASHA per habitation, dependant on workload etc.
2. The States will also need to work out the district and block-wise coverage/phasing for selection of ASHAs.
3. It is envisaged that the selection and training process of ASHA will be given due attention

by the concerned State to ensure that at least 40 percent of the envisaged.

4. ASHAs in the State are selected and given induction training in the first year as per the norms given in the guidelines. Rest of the ASHAs can subsequently be selected and trained during second and third year.

Criteria for Selection: ASHA must be a woman resident of the village, preferably in the age group of 25 to 45 yrs, with effective communication skills, leadership qualities and be able to reach out to the community. She should be literate with formal education up to Eighth Class. This may be relaxed only if no suitable person with this qualification is available. Adequate representation from disadvantaged population groups should be ensured to serve such groups better.

TRAINING

After selection, ASHA will have to undergo series of training episodes to acquire the necessary knowledge, skills and confidence for performing her spelled out roles. Considering range of functions and tasks to be performed, induction training may be completed in 23 days spread over a period of 12 months. The first round may be of seven days, to be followed by another four rounds of training, each lasting for four days to complete induction training.

Training materials: would be prepared according to the roles and responsibilities that the ASHA would need to perform. Her envisaged functions and tasks will be expanded into a listing of competencies and the training material would be prepared accordingly. The training materials produced at the national level would be in the form of a general prototype, which states may modify and adapt as per local needs. The training material will include facilitator's guide, training aids and resource material for ASHAs.

Periodic Trainings: After the induction training, periodic re-training will be held for about two days, once in every alternate month at appropriate level for all ASHAs. During this training, interactive sessions will be held to help refresh and upgrade their knowledge and skills, trouble shoot problems they are facing, monitor their work and also for keeping up motivation and interest. The opportunity will also be used for replenishments of supplies and payment of performance linked incentives. ASHAs will be compensated for attending these meetings.

On-the-job Training : ASHAs needs to have on the job support after training both during the initial training phase and during the later periodic training phase it is needed to provide on the job training to ASHAs in the field, so that they can get individual attention and support that is essential to begin and continue her work. ANMs while conducting outreach sessions in the villages will contact ASHA of the village and use the opportunity for continuing education. NGOs can also be invited to take up the selection; training and post training follow up. Similarly block facilitators identified earlier for selection of ASHAs can also be engaged for regular field support.

Continuing Education and skill upgradation: A resource agency in the district of state (preferably an NGO) will be identified by the State. The resource agency in collaboration with open schools and other appropriate community health distance education schemes will develop relevant illustrated material to be mailed to ASHAs periodically for those who would opt for an eventual certification.

ROLE AND INTEGRATION WITH ANGANWADI

(a) Anganwadi Worker (AWW) will Guide ASHA in performing following activities: Organizing Health Day once/twice a month. On health day, the women, adolescent girls and children from the village will be mobilized for orientation on health related issues such as importance of nutritious food, personal hygiene, care during pregnancy, importance of antenatal check up and institutional delivery, home remedies for minor ailment and importance of immunization etc. AWWs will inform ANM to participate & guide organizing the Health Days at Anganwadi Centre (AWC).

(b) AWWs and ANMs will act as a resource persons for the training of ASHA.

(c) IEC activity through display of posters, folk dances etc. on these days can be undertaken to sensitize the beneficiaries on health related issues.

(d) Anganwadi worker will be depot holder for drug kits and will be issuing it to ASHA. The replacement of the consumed drugs can also be done through AWW.

(e) AWW will update the list of eligible couples and also the children less than one year of age in the village with the help of ASHA.

(f) ASHA will support the AWW in mobilizing pregnant and lactating women and infants for nutrition supplement. She would also take initiative for bringing the beneficiaries from the village on specific days of immunization. health checkups / health days etc. to Anganwadi Centres.

ROLE AND INTEGRATION WITH ANM

Auxiliary Nurse Midwife (ANM) will Guide ASHA in performing following activities:

1. She will hold weekly / fortnightly meeting with ASHA and discuss the activities undertaken during the week / fortnight. She will guide her in case ASHA had encountered any problem during the performance of her activity.
2. AWWs and ANMs will act as a resource person for the training of ASHA.
3. ANMs will inform ASHA regarding date and time of the outreach session and will also guide her for bringing the beneficiary to the outreach session.
4. ANM will participate & guide in organizing the Health Days at AWC.
5. She will take help of ASHA in updating eligible couple register of the village concerned.
6. She will utilize ASHA in motivating the pregnant women for coming to sub centre for initial checkups. She will also help ANMs in bringing married couples to sub centres for adopting family planning.
7. ANM will guide ASHA in motivating pregnant women for taking full course of IFA Tablets and TT Injections etc.
8. ANMs will orient ASHA on the dose schedule and side affects of oral pills.
9. ANMs will educate ASHA on danger signs of pregnancy and labour so that she can timely identify and help beneficiary in getting further treatment.
10. ANMs will inform ASHA on date, time and place for initial and periodic training schedule. She will also ensure that during the training ASHA gets the compensation for performance and also TA/DA for attending the training.

COMPENSATION TO ASHA:

ASHA would be an honorary volunteer and would not receive any salary or honorarium. Her work would be so tailored that it does not interfere with her normal livelihood. However ASHA could be compensated for her time in the following situations:

a) For the duration of her training both in terms of TA and DA. (So that her loss of livelihood for those days is partly compensated)

b) For participating in the monthly/bi-monthly training, as the case may be. (For situations (a) and (b), payment will be made at the venue of the training when ASHAs come for regular training sessions and meetings).

c) Wherever compensation has been provided for under different national programmes for undertaking specific health or other social sector programmes with measurable outputs, such tasks should be assigned to ASHAs on priority (i.e. before it is offered to other village volunteers) wherever they are in position. (For situation (c) disbursement of compensation to ASHAs will be made as per the specific payment mechanism built into individual programmes).

d) Other than the above specific programmes, a number of key health related activities and service outcomes are aimed within a village (For example all eligible children immunized, all newborns weighed, all pregnant women attended an antenatal clinic etc). The Untied Fund of Rs.10,000/- at the Sub-centre level (to be jointly operated by the ANM and the Sarpanch) could be used as monetary compensation to ASHA for achieving these key processes. The exact package of processes that form the package would be determined at the state level depending on the supply-side constraints and what is feasible to achieve within the specified time period.

MONITORING AND EVALUATION: GOI has set up following indicators for monitoring ASHA.

Process Indicators:

(a) Number of ASHAs selected by due process
(b) Number of ASHAs trained
(c) % of ASHAs attending review meetings after one year

Outcome Indicators:

(a) % of newborn who were weighed and families counseled
(b) % of children with diarrhoea who received ORS
(c) % of deliveries with skilled assistance
(d) % of institutional deliveries
(e) % of JSY claims made to ASHA
(f) % completely immunized in 12-23 months age group
(g) % of unmet need for spacing contraception among BPL
(h) % of fever cases who received chloroquine within first week in an malaria endemic area

Impact indicators:

(a) IMR
(b) Child malnutrition rates
(c) Number of cases of TB/leprosy cases detected as compared to previous year.

10. Janani Surksha Yojana (JSY)

Introduction

Janani Suraksha Yojana (JSY) under the overall umbrella of National Rural Health Mission (NRHM) is proposed as a modification of the existing National Maternity Benefit Scheme (NMBS). While NMBS is linked to provision of better diet for pregnant women from BPL families, JSY integrates the cash assistance with antenatal care during the pregnancy period, institutional care during delivery and immediate post-partum period in a health centre by establishing a system of coordinated care by ?eld level health worker. The JSY would be a 100% centrally sponsored scheme.

Goals

- To reduce over all maternal mortality ratio and infant mortality rate
- To increase institutional deliveries in BPL families.

Beneficiaries

All pregnant women belonging to the below poverty line (BPL) households who are 19 years or above. The support will be only up to two live births.

1. The beneits would be extended to all women from BPL families of 10 low performing states namely 8 EAG states (Uttar Pradesh, Uttaranchal, Madhya Pradesh, Chhattisgarh, Rajasthan, Bihar, Jharkhand and Orissa) and the states of Assam and J&K even after the third live birth if the mother, of her own accord chooses to undergo sterilization in the health facility where she delivered, immediately after the delivery. Satisfaction of the Medical officer through a process, about the number of living children of the expectant mother would be a pre-condition to availing the bene?t of this scheme.
2. The beneits would also be available to such pregnant women falling in the above category even though not registered under JSY previously during pregnancy period but needing institutional care for delivery including management of complications like obstructed labour, PPH, eclampsia, PP sepsis etc. State will devise necessary mechanisms for adequate certi?cation from the Medical oficer of the health institution from where woman has taken treatment. This would be essential for disbursement of beneit.

Cash Benefit

Gateory of States	Rural Area			Urban Area		
	Assistance Package to mother	Package for the Accredited Worker	Total	Assistance Package to mother	Package for the Accredited Worker	Total
LPS	700	600	1300	600	200	800
HPS	700	NIL	700			

LPS: Low performing states; HPS: High performing states

Cash incentive to the ASHA: ASHA would receive a cash incentive of Rs.200/- per delivery case facilitated by her. The Assistance package to the ASHA or an equivalent worker is available only if she works and assists the pregnant women. If any pregnant women does not take assistance of any accredited worker, may be because no ASHA is in position, she should be paid the sum total of both the packages.

Assistance for Caesarean Section : FRUs/CHCs would provide emergency obstetric services. Where Government specialists are not available in a health institution, assistance up to Rs. 1500/- per case will be provided for hiring services of private experts to carry out the surgery either in a Government medical facility or in Private hospital, nursing home, etc.

Compensation payment for Tubectomy/ Laparoscopy: If hospitalization for delivery is followed immediately by Tubectomy/laparoscopy, compensation money available under the Family welfare scheme would also be paid to JSY beneficiary in the health centre as per the existing procedure followed for payment of compensation money.

Role of Registered Accredited Worker/ASHA

ASHA or an equivalent worker under supervision of ANM/AWW would have the following role:

1. Identify pregnant woman from BPL families as a beneficiary of the scheme
2. Report to the ANM and bring the women to the sub-centre/PHC for registration
3. Assist the woman to obtain BPL certification if BPL card is not available
4. Provide and / or help the women to receive at least three ANC
5. Counsel for institutional delivery and fix before 7th month of pregnancy the place of delivery, in close consultation with the ANM and the PHC and inform the beneficiary
6. Assist in receiving two TT injection
7. When the pregnant woman is in labour or faces complication, escort the women to the pre-determined health centre and stay with her till the delivery is complete and woman is discharged.
8. Arrange to immunize the newborn till the age of 10 weeks
9. Register birth or death of the child or mother
10. Post natal visits within 7 days of pregnancy and track mother's health
11. Counsel for initiation of breastfeeding within one-hour of delivery and its continuance till 3-6 months.

Drawing a micro-birth plan for each beneficiary

Sr. No.	Acitivity	To be undertaken by	Proposed Time Line
a)	Identification of beneficiary and filling up of the JSY card. (See ANNEXURE-V)	ASHA or an equivalent worker (Those registered with SC/PHC)	Atleast 16-20 weeks before the expected date of delivery.
b)	Registering the expectant mother for ANC in the sub-acute/health centre. Filling of Maternal and child card (which will be part of the JSY Card.)	Same as above Registered accrediated worker should be present during registration.	To start immediately on identification
c)	Preparing the birth plan including dates of ANCs and recording it on the JSY card and inform the mother.	ANH in the presence of ASHA possibly in consultation with husband or other family members.	At least 8-10 weeks before the expected date of delivery.
d)	Completion of formalities for receiving JSY benefit Including collecting necessary BPL certificates wherever necessary from Panchayat/ local bodies/Municipalities	Registered ASHA or an equivalent worker	Within 2-3 weeks from identification
e)	Motivating for institutional delivery by explaining enhanced JSY benefits.	ASHA or an equivalent worker in consultation with MO, PHC	Within 2-3 weeks of identification.
f)	Identify the health centre for all referrral as well as the place of delivery and inform the pregnant women/her husband/family member and the Registered ASHA.		
g)	Submit the completed JSY card in the Health centre for verification by the authorized / Medical officer. Taking necessary steps towards delivery as well as to make available fund to ANM/Health worker/ASHA etc.	MO, PHC	Before 2 weeks of expected date of delivery

Monitoring and Evaluation

There will be a mandatory meeting of all accredited worker on the third Friday of every month, at the sub-centre. If Friday is a holiday, meeting should be held on following working day. In the Friday meeting, the ANM will prepare a Monthly Work.

Schedule of each village level worker:

1. Possible number of pregnant women under JSY to be taken to the health centre/Anganwadi for ANC.
2. Possible number of pregnant women registered under JSY to be taken to the health centre for delivery.
3. Possible number of children/pregnant women to be taken to the health centre/Anganwadi for immunization.
4. Ensure that the compensation, incentive and referral money is ready for disbursement and the due official process has been set in motion.
5. Feed back on following points should be taken: (a) number of children immunized (b) number of pregnant women visited (c) number of post natal visits and (d) cases referred in the month.

11. National Rural Health Mission (2005-2012)

Preamble

Recognizing the importance of Health in the process of economic and social development and improving the quality of life of our citizens, the Government of India has resolved to launch the National Rural Health Mission to carry out necessary architectural correction in the basic health care delivery system.

The Mission adopts a synergistic approach by relating health to determinants of good health viz. segments of nutrition, sanitation, hygiene and safe drinking water. It also aims at mainstreaming the Indian systems of medicine to facilitate health care.

The Plan of Action includes increasing public expenditure on health, reducing regional imbalance in health infrastructure, pooling resources, integration of organizational structures, optimization of health manpower, decentralization and district management of health programmes, community participation and ownership of assets, induction of management and financial personnel into district health system, and operationalizing community health centers into functional hospitals meeting Indian Public Health Standards in each Block of the Country.

The Goal of the Mission is to improve the availability of and access to quality health care by people, especially for those residing in rural areas, the poor, women and children.

NRHM – The Vision (objectives)

1. The NRHM seeks to provide effective healthcare to rural population throughout the country with special focus on 18 states, which have weak public health indicators and/or weak infrastructure. Viz.
   - Arunachal Pradesh, Assam
   - Bihar
   - Chattisgarh
   - Himachal Pradesh
   - Jharkhand, Jammu & Kashmir
   - Manipur, Mizoram, Meghalaya, Madhya Pradesh
   - Nagaland
   - Orissa
   - Rajasthan
   - Sikkim
   - Tripura
   - Uttaranchal and Uttar Pradesh
2. Raise public spending on Health from 0.9% of GDP to 2-3% of GDP
3. Undertake architectural correction of the health system to enable it to effectively handle increased allocations under the National Common Minimum Programme
4. Key components are:
   i. Female health activist (*Accredited Social Health Activist* – ASHA), in each village
   ii. Village health plan prepared through a local team headed by the Health & Sanitation Committee of the Panchayat
   iii. Strengthening of the rural hospital for effective curative care and made measurable and accountable to the community through *Indian Public Health Standards* (IPHS)
   iv. Integration of vertical Health & Family Welfare Programmes and Funds for optimal utilization of funds and infrastructure and strengthening delivery of primary healthcare.
5. Revitalize local health traditions and mainstream Ayurveda, Yoga, Unani, Siddha and Homeopathy (AYUSH) into the public health system
6. Effective integration of health concerns with determinants of health like sanitation & hygiene, nutrition, and safe drinking water through a District Plan for Health
7. Decentralization of programmes for district management of health
8. Address the inter-state and inter-district disparities, especially among the 18 high focus States, including unmet needs for public health infrastructure.
9. Define time-bound goals and report publicly on their progress.
10. Improve access of rural people, especially poor women and children, to equitable, affordable, accountable and effective primary healthcare.

Goals

1. Reduction in Infant Mortality Rate (IMR) and Maternal Mortality Ratio (MMR)
2. Universal access to public health services such as Women's health, child health, water, sanitation & hygiene, immunization, and Nutrition.
3. Prevention and control of communicable and non-communicable diseases, including locally endemic diseases
4. Access to integrated comprehensive primary healthcare
5. Population stabilization, gender and demographic balance.
6. Revitalize local health traditions and mainstream AYUSH
7. Promotion of healthy life styles

Strategies

Core Strategies

1. Train and enhance capacity of Panchayati Raj Institutions (PRIs) to own, control and manage public health services.
2. Promote access to improved healthcare at household level through the female health activist (ASHA).
3. Health Plan for each village through Village Health Committee of the Panchayat
4. Strengthening sub-centre through an untied fund to enable local planning and action and more Multi Purpose Workers (MPWs)
5. Strengthening existing PHCs and CHCs, and provision of 30-50 bedded CHC per lakh population for improved curative care to a normative standard (Indian Public Health Standards defining personnel, equipment and management standards)
6. Preparation and Implementation of an inter-sectoral 'District Health Plan' prepared by the District Health Mission, including drinking water, sanitation & hygiene and nutrition.
7. Integrating vertical Health and Family Welfare programmes at National, State, Block, and District levels.
8. Technical Support to National, State and District Health Missions, for Public Health Management
9. Strengthening capacities for data collection, assessment and review for evidence based planning, monitoring and supervision.
10. Formulation of transparent policies for deployment and career development of Human Resources for health
11. Developing capacities for preventive health care at all levels for promoting healthy life styles, reduction in consumption of tobacco and alcohol etc.
12. Promoting non-profit sector particularly in under served areas.

Supplementary Strategies

1. Regulation of Private Sector including the informal rural practitioners to ensure availability of quality service to citizens at reasonable cost
2. Promotion of Public Private Partnerships for achieving public health goals
3. Mainstreaming AYUSH – revitalizing local health traditions
4. Reorienting medical education to support rural health issues including regulation of Medical care and Medical Ethics
5. Effective and viable risk pooling and social health insurance to provide health security to the poor by ensuring accessible, affordable, accountable and good quality hospital care.

Plan of Action

A) Accredited Social Health Activists (ASHA)

1. One per village - chosen by and accountable to the panchayat
2. Honorary volunteer, receiving performance-based compensation for promoting universal immunization, referral and escort services for RCH, construction of household toilets, and other healthcare delivery programmes
3. Will be trained on pedagogy of public health developed and mentored through 'Standing Mentoring Group' at National level incorporating best practices and implemented through active involvement of community health resource organizations
4. Will be given a Drug Kit containing generic AYUSH and allopathic formulations for common ailments. The drug kit would be replenished from time to time.
5. Induction training of ASHA to be of 23 days in all, spread over 12 months, on job training would continue throughout the year
6. Government of India will bear the cost of training, incentives and medical kits. The

remaining components will be funded under Financial Envelope given to the States under the programme

7. Prototype training material to be developed at National level subject to State level modifications
8. Cascade model of training proposed through Training of Trainers including contract plus distance learning model
9. Training would require partnership with NGOs/ICDS Training Centres and State Health Institutes
10. Would act as a bridge between the ANM (Public Health System) and the village
11. Will facilitate preparation and implementation of the Village Health Plan along with Anganwadi worker, ANM, functionaries of other Departments, and Self Help Group members, under the leadership of the Village Health Committee of the Panchayat.

B) Strengthening Sub-Centres

1. Each sub-centre will have an Untied Fund for local action @ Rs. 10,000 per annum. This Fund will be deposited in a joint Bank Account of the ANM & Sarpanch and operated by the ANM, in consultation with the Village Health Committee
2. Supply of essential drugs, both allopathic and AYUSH, to the Sub-centres
3. In case of additional Outlays, Multipurpose Workers (Male)/Additional ANMs wherever needed, sanction of new Sub-centres as per 2001 population norm, and upgrading existing Sub-centres, including buildings for Sub-centres functioning in rented premises will be considered

C) Strengthening Primary Health Centres

Mission aims at Strengthening PHC for quality preventive, promotive, curative, supervisory and Outreach services, through:

1. Adequate and regular supply of essential quality drugs and equipment (including Supply of Auto Disabled Syringes for immunization) to PHCs.
2. Provision of 24 hour service in 50% PHCs by addressing shortage of doctors, especially in high focus States, through mainstreaming AYUSH manpower.
3. Observance of Standard treatment guidelines & protocols.
4. In case of additional Outlays, intensification of ongoing communicable disease control programmes, new programmes for control of non-communicable diseases, upgradation of 100% PHCs for 24 hours referral service, and provision of 2nd doctor at PHC level (I male, 1 female) would be undertaken on the basis of felt need.

D) Strengthening CHCs For First Referral Care

A key strategy of the Mission is:

1. Operationalizing 3222 existing Community Health Centres (30-50 beds) as 24 Hour First Referral Units, including posting of anesthetists.
2. Codification of new Indian Public Health Standards, setting norms for infrastructure, staff, equipment, management etc. for CHCs.
3. Promotion of Stakeholder Committees (Rogi Kalyan Samitis) for hospital management.
4. Developing standards of services and costs in hospital care.
5. Develop, display and ensure compliance to Citizen's Charter at CHC/PHC level.
6. In case of additional Outlays, creation of new Community Health Centres (30-50 beds) to meet the population norm as per Census 2001, and bearing their recurring costs for the Mission period could be considered.

E) District Health Plan

1. District Health Plan would be an amalgamation of field responses through Village Health Plans, State and National priorities for Health, Water Supply, Sanitation and Nutrition
2. Health Plans would form the core unit of action proposed in areas like water supply, sanitation, hygiene and nutrition. Implementing Departments would integrate into District Health Mission for monitoring.
3. District becomes core unit of planning, budgeting and implementation.
4. Centrally Sponsored Schemes could be rationalized/modified accordingly in consultation with States.
5. Concept of "funneling" funds to district for effective integration of programmes.
6. All vertical Health and Family Welfare Programmes at District and state level merge into one common "District Health Mission" at the District level and the "State Health Mission" at the state level.

7. Provision of Project Management Unit for all districts, through contractual engagement of MBA, Inter Charter/Inter Cost and Data Entry Operator, for improved programme management.

F) Converging Sanitation and Hygiene under NRHM

1. Total Sanitation Campaign (TSC) is presently implemented in 350 districts, and is proposed to cover all districts in 10th Plan.
2. Components of TSC include IEC activities, rural sanitary marts, individual household toilets, women sanitary complex, and School Sanitation Programme.
3. Similar to the DHM, the TSC is also implemented through Panchayati Raj Institutions (PRIs).
4. The District Health Mission would therefore guide activities of sanitation at district level, and promote joint IEC for public health, sanitation and hygiene, through Village Health & Sanitation Committee, and promote household toilets and School Sanitation Programme. The Mission would incentivize ASHA for promoting household toilets.

G) Strengthening Disease Control Programmes

1. National Disease Control Programmes for Malaria, TB, Kala Azar, Filaria, Blindness & Iodine Deficiency and Integrated Disease Surveillance Programme shall be integrated under the Mission, for improved programme delivery.
2. New Initiatives would be launched for control of Non Communicable Diseases.
3. Disease surveillance system at village level would be strengthened.
4. Supply of generic drugs (both AYUSH & Allopathic) for common ailments at village, SC, PHC/CHC level.
5. Provision of a mobile medical unit at District level for improved Outreach services.

H) Public-Private Partnership For Public Health Goals, Including Regulation Of Private Sector

1. Since almost 75% of health services are being currently provided by the private sector, there is a need to refine regulation.
2. Regulation to be transparent and accountable
3. Reform of regulatory bodies/creation where necessary.
4. District Institutional Mechanism for Mission must have representation of private sector.
5. Need to develop guidelines for Public-Private Partnership (PPP) in health sector. Identifying areas of partnership, which are need based, thematic and geographic.
6. Public sector to play the lead role in defining the framework and sustaining the partnership
7. Management plan for PPP initiatives: at District/State and National levels.

I) New Health Financing Mechanisms

A Task Group to examine new health financing mechanisms, including Risk Pooling for Hospital Care as follows:

1. Progressively the District Health Missions to move towards paying hospitals for services by way of reimbursement, on the principle of "money follows the patient".
2. Standardization of services – outpatient, in-patient, laboratory, surgical interventions- and costs will be done periodically by a committee of experts in each state.
3. A National Expert Group to monitor these standards and give suitable advice and guidance on protocols and cost comparisons.
4. All existing CHCs to have wage component paid on monthly basis. Other recurrent costs may be reimbursed for services rendered from District Health Fund. Over the Mission period, the CHC may move towards all costs, including wages reimbursed for services rendered.
5. A district health accounting system, and an ombudsman to be created to monitor the District Health Fund Management, and take corrective action.
6. Adequate technical managerial and accounting support to be provided to DHM in managing risk-pooling and health security.
7. Where credible Community Based Health Insurance Schemes (CBHI) exist/are launched, they will be encouraged as part of the Mission.
8. The Central government will provide subsidies to cover a part of the premiums for the poor, and monitor the schemes.
9. The IRDA will be approached to promote such CBHIs, which will be periodically evaluated for effective delivery.

J) Reorienting Health/Medical Education To Support Rural Health Issues

1. While district and tertiary hospitals are necessarily located in urban centres, they form an integral part of the referral care chain serving the needs of the rural people.
2. Medical and para-medical education facilities need to be created in states, based on need assessment.
3. Suggestion for Commission for Excellence in Health Care (Medical Grants Commission), National Institution for Public Health Management etc.
4. Task Group to improve guidelines/details

Institutional Mechanisms

1. Village Health & Sanitation Samiti (at village level consisting of Panchayat Representative/ s, ANM/MPW, Anganwadi worker, teacher, ASHA, community health volunteers.
2. Rogi Kalyan Samiti (or equivalent) for community management of public hospitals.
3. District Health Mission, under the leadership of Zila Parishad with District Health Head as Convener and all relevant departments, NGOs, private professionals etc represented on it.
4. State Health Mission, Chaired by Chief Minister and co-chaired by Health Minister and with the State Health Secretary as Convener-representation of related departments, NGOs, private professionals etc.
5. Integration of Departments of Health and Family Welfare, at National and State level.
6. National Mission Steering Group chaired by Union Minister for Health & Family Welfare with Deputy Chairman Planning Commission, Ministers of Panchayat Raj, Rural Development and Human Resource Development and public health professionals as members, to provide policy support and guidance to the Mission.
7. Empowered Programme Committee chaired by Secretary HFW, to be the Executive Body of the Mission.
8. Standing Mentoring Group shall guide and oversee the implementation of ASHA initiative.
9. Task Groups for Selected Tasks (time-bound).

Technical Support

1. To be effective the Mission needs a strong component of Technical Support
2. This would include reorientation into public health management.
3. Reposition existing health resource institutions, like Population Research Centre (PRC), Regional Resource Centre (RRC), State Institute of Health & Family Welfare (SIHFW).
4. Involve NGOs as resource organisations.
5. Improved Health Information System.
6. Support required at all levels: National, State, District and sub-district.
7. Mission would require two distinct support mechanisms – a) Program Management Support Centre and b) Health Trust of India.

a) Program Management Support Centre

1. For Strengthening Management Systems-basic program management, financial systems, infrastructure maintenance, procurement & logistics systems, Monitoring & Information System (MIS), non-lapsable health pool etc.
2. For Developing Manpower Systems – recruitment (induction of MBAs/CAs /MCAs), training & curriculum development (revitalization of existing institutions & partnerships with NGO & private sector. Sector institutions), motivation & performance appraisal etc.
3. For Improved Governance – decentralization & empowerment of communities, induction of IT based systems like e-banking, social audit and right to information.

b) Health Trust Of India

1. Proposed as a knowledge institution, to be the repository of innovation – research & documentation, health information system, planning, monitoring & evaluation etc.
2. For establishing Public Accountability Systems – external evaluations, community based feedback mechanisms, participation of PRIs / NGOs etc.
3. For developing a Framework for pro-poor Innovations.
4. For reviewing Health Legislations.
5. A base for encouraging experimentation and action research.
6. For inter & intra Sector Networking with National and International Organizations.
7. Think Tank for developing a long-term vision of the Sector & for building planning capacities of PRIs, Districts etc.

Role of State Governments Under NRHM

1. The Mission covers the entire country. The 18 high focus States are Uttar Pradesh, Bihar, Rajasthan, Madhya Pradesh, Orissa, Uttaranchal, Jharkhand, Chattisgarh, Assam, Sikkim, Arunachal Pradesh, Manipur, Meghalaya, Tripura, Nagaland, Mizoram Himachal Pradesh and Jammu & Kashmir. GOI would provide funding for key components in these 18 high focus States. Other States would fund interventions like ASHA, Programme Management Unit (PMU), and upgradation of SC/PHC/CHC through Integrated Financial Envelope.
2. NRHM provides broad conceptual framework. States would project operational modalities in their State Action Plans, to be decided in consultation with the Mission Steering Group.
3. NRHM would prioritize funding for addressing inter-state and intradistrict disparities in terms of health infrastructure and indicators.
4. States would sign Memorandum of Understanding with Government of India, indicating their commitment to increase contribution to Public Health Budget (preferably by 10% each year), increased devolution to Panchayati Raj Institutions as per 73rd Constitution (Amendment) Act, and performance benchmarks for release of funds.

Focus on The North Eastern States

1. All 8 North East States, including Assam, Arunachal Pradesh, Manipur, Meghalaya, Mizoram, Nagaland, Sikkim and Tripura, are among the States selected under the Mission, for special focus.
2. Empowerment to the Mission would mean greater flexibilities for the 10% committed Outlay of the Ministry of Health & Family Welfare, for North East States.
3. States shall be supported for creation/ upgradation of health infrastructure, increased mobility, contractual engagement, and technical support under the Mission.
4. Regional Resource Centre is being supported under NRHM for the North Eastern States.
5. Funding would be available to address local health issues in a comprehensive manner, through State specific schemes and initiatives.

Role of Panchayati Raj Institutions

The Mission envisages the following roles for PRIs

1. States to indicate in their MoUs the commitment for devolution of funds, functionaries and programmes for health, to PRIs.
2. The District Health Mission to be led by the Zila Parishad. The DHM will control, guide and manage all public health institutions in the district, Sub-centres, PHCs and CHCs.
3. ASHAs would be selected by and be accountable to the Village Panchayat.
4. The Village Health Committee of the Panchayat would prepare the Village Health Plan, and promote intersectoral integration
5. Each sub-centre will have an Untied Fund for local action @ Rs. 10,000 per annum. This Fund will be deposited in a joint Bank Account of the ANM & Sarpanch and operated by the ANM, in consultation with the Village Health Committee.
6. PRI involvement in Rogi Kalyan Samitis for good hospital management.
7. Provision of training to members of PRIs
8. Making available health related databases to all stakeholders, including Panchayats at all levels.

Role of NGOs In The Mission

1. Included in institutional arrangement at National, State and District levels, including Standing Mentoring Group for ASHA
2. Member of Task Groups
3. Provision of Training, BCC and Technical Support for ASHAs/DHM
4. Health Resource Organizations
5. Service delivery for identified population groups on select themes
6. For monitoring, evaluation and social audit

Mainstreaming AYUSH

1. The Mission seeks to revitalize local health traditions and mainstream AYUSH infrastructure, including manpower, and drugs, to strengthen the public health system at all levels.
2. AYUSH medications shall be included in the Drug Kit provided at village levels to ASHA
3. The additional supply of generic drugs for common ailments at Subcentre/ PHC/CHC levels under the Mission shall also include AYUSH formulations.

4. At the CHC level, two rooms shall be provided for AYUSH practitioner and pharmacist under the Indian Public Health System (IPHS) model
5. Single doctor PHCs shall be upgraded to two doctor PHCs by mainstreaming AYUSH practitioner at that level.

Funding Arrangements

1. The Mission is conceived as an umbrella programme subsuming the existing programmes of health and family welfare, including the RCHII, National Disease Control Programmes for Malaria, TB, Kala Azar, Filaria, Blindness & Iodine Deficiency and Integrated Disease Surveillance Programme.
2. The Budget Head For NRHM shall be created in B.E. 2006-07 at National and State levels. Initially, the vertical health and family welfare programmes shall retain their Sub-Budget Head under the NRHM.
3. The Outlay of the NRHM for 2005-06 is in the range of Rs.6700 crores.
4. The Mission envisages an additionality of 30% over existing Annual Budgetary Outlays, every year, to fulfill the mandate of the National Common Minimum Programme to raise the Outlays for Public Health from 0.9% of GDP to 2-3% of GDP.
5. The Outlay for NRHM shall accordingly be determined in the Annual Budgetary exercise.
6. The States are expected to raise their contributions to Public Health Budget by minimum 10% p.a. to support the Mission activities.
7. Funds shall be released to States through SCOVA, largely in the form of Financial Envelopes, with weightage to 18 high focus States.

Timelines (For Major Components)

- Merger of Multiple Societies June 2005.
- Constitution of District/State Mission Provision of additional generic drugs at SC/PHC/CHC level December 2005.
- Operational Programme Management Units 2005-2006.
- Preparation of Village Health Plans 2006.
- ASHA at village level (with Drug Kit) 2005-2008.
- Upgrading of Rural Hospitals 2005-2007.
- Operationalizing District Planning 2005-2007.
- Mobile Medical Unit at district level 2005-08.

Outcomes

(a) National Level

1. Infant Mortality Rate reduced to 30/1000 live births.
2. Maternal Mortality Ratio reduced to 100/ 100,000.
3. Total Fertility Rate reduced to 2.1.
4. Malaria mortality reduction rate –50% upto 2010, additional 10% by 2012.
5. Kala Azar mortality reduction rate: 100% by 2010 and sustaining elimination until 2012.
6. Filaria/Microfilaria reduction rate: 70% by 2010, 80% by 2012 and elimination by 2015
7. Dengue mortality reduction rate: 50% by 2010 and sustaining at that level until 2012.
8. Japanese Encephalitis mortality reduction rate: 50% by 2010 and sustaining at that level until 2012.
9. Cataract Operation: increasing to 46 lakhs per year until 2012.
10. Leprosy prevalence rate: reduce from 1.8/ 10,000 in 2005 to less than 1/10,000 thereafter.
11. Tuberculosis DOTS services: Maintain 85% cure rate through entire Mission period.
12. Upgrading Community Health Centers to Indian Public Health Standards.
13. Increase utilization of First Referral Units from less than 20% to 75%.
14. Engaging 250,000 female Accredited Social Health Activists (ASHAs) in 10 States.

(b) Community Level

1. Availability of trained community level worker at village level, with a drug kit for generic ailments.
2. Health Day at Anganwadi level on a fixed day/ month for provision of immunization, ante/ post natal checkups and services related to mother & child healthcare, including nutrition.
3. Availability of generic drugs for common ailments at Sub-centre and hospital level.
4. Good hospital care through assured availability of doctors, drugs and quality services at PHC/ CHC level.
5. Improved access to Universal Immunization through induction of Auto Disabled Syringes, alternate vaccine delivery and improved mobilization services under the programme.

6. Improved facilities for institutional delivery through provision of referral, transport, escort and improved hospital care subsidized under the Janani Suraksha Yojana (JSY) for the Below Poverty Line families.
7. Availability of assured healthcare at reduced financial risk through pilots of Community Health Insurance under the Mission.
8. Provision of household toilets.
9. Improved Outreach services through mobile medical unit at districtlevel.

Monitoring and Evaluation

1. Health MIS to be developed upto CHC level, and web-enabled for citizen scrutiny
2. Sub-centres to report on performance to Panchayats, Hospitals to Rogi Kalyan Samitis and District Health Mission to Zila Parishad
3. The District Health Mission to monitor compliance to Citizen's Charter at CHC level
4. Annual District Reports on People's Health (to be prepared by Govt/NGO collaboration)
5. State and National Reports on People's Health to be tabled in Assemblies, Parliament.
6. External evaluation/social audit through professional bodies/NGOs
7. Mid Course reviews and appropriate correction.

12. World Health Day 2008 : Protecting Health From Climate Change

BACKGROUND

Climate change: an issue for the health sector

Health hazards from climate change are diverse and global in nature. The hazards range from higher risks of extreme weather events to changes in the dynamics of infectious diseases. Many of the leading killer diseases are sensitive to climatic conditions; their incidence and spread are likely to be affected by changing weather patterns. The health impacts of climate change are already evident in different ways: more people are dying from excessive heat than before, changes are occurring in the incidence of vector-borne diseases, and the pattern of natural disasters is altering. These impacts will be disproportionately greater in vulnerable populations, which include the very young, elderly, medically infirm, poor and isolated populations. Vulnerability is also high in:

1. Areas with a high endemicity of climate-sensitive diseases, severe water scarcity, and low food production;
2. Small-island developing states and mountainous regions; and
3. Megacities and coastal areas in developing countries.

Action needs to be taken now

The health impacts of climate change will be difficult to reverse in a few years or decades. Yet, many of these possible impacts can be avoided or controlled. There are established steps in health and related sectors to reduce the exposure to and the effect of changing climate. For example, controlling disease vectors, reducing pollution from transport, and efficient land use and water management are well-known and tested measures that can help. Moreover, many of the steps needed to prevent climate change have positive health benefits. For example, increased use of bicycles and public transport instead of personal cars in industrialized countries will reduce greenhouse gas emissions. It will also improve air quality and lead to better respiratory health and fewer premature deaths. The increase in physical activity from cycling and walking may lead to less obesity and fewer obesity-related illness. The sooner these steps are taken, the greater their impact will be on public health.

Aims and objectives of World Health Day 2008

The objective of World Health Day 2008 is to catalyse public participation in the global campaign to protect health from the adverse effects of climate change. WHO aims to put public health at the centre of the UN agenda on climate change. This is an opportunity for the international agencies, nongovernmental organizations, and governments as well as WHO to :

1. Establish links between climate change and health and other development areas such as environment, food, energy, transport;
2. Hold events/activities in countries to publicize issues related to the impact of climate change on health;
3. Involve as wide a spectrum of the world population as possible in efforts to stabilize climate change;
4. Create advocacy campaigns for generating momentum that compels governments, the international community, civil society and individuals to take action;
5. Protect poor and vulnerable populations from the effects of climate change, especially in Africa.

Goals for World Health Day 2008

1. Raise awareness and public understanding of the global and locally relevant health consequences of climate change.
2. Advocate for interdisciplinary and intersectoral partnerships from the local to international level that seek to improve health through rapid deployment of mitigation strategies to stabilize climate change and development of proactive adaptation programmes to minimize health impacts.
3. Generate effective actions by local communities, organizations, health systems and governments to reduce the impact of climate change on health through urgent application of mitigation and adaptation techniques.
4. Demonstrate the health community's role in facing the challenges globally and in regions, countries and communities.

5. Spark commitment and action among governments, international organizations, donors, civil society, businesses and communities (especially among young people) to anchor health at the heart of the climate change agenda.

KEY MESSAGES FOR WORLD HEALTH DAY 2008

Health is one of the areas most affected by climate change – and it is being affected now

The science is clear. The earth is warming, the warming is accelerating, and human actions are responsible. If current warming trends remain uncontrolled, humanity will face more injury, disease and death related to natural disasters and heatwaves; higher rates of foodborne, waterborne, and vector-borne illness; and more premature deaths and disease related to air pollution. Moreover, in many parts of the world, large populations will be displaced by rising sea level and affected by drought and famine. As glaciers melt, the hydrological cycle shifts and the productivity of arable land changes. We are beginning to be able to measure some of these effects on health even now.

The health impacts of climate change will hit the poor hardest

The physical effects of climate change will vary in different geographical locations. The human health impacts from climate change are further modified by such conditions as level of development, poverty and education, public health infrastructure, land use practices and political structure. Initially, developing countries will be hit the hardest. Countries with high levels of poverty and malnutrition, weak health infrastructures and/or political unrest will be the least able to cope. Moreover, if we fail to address climate change and its effects on health, we risk jeopardizing even further our ability to achieve the Millennium Development Goals.

Traditional public health tools are important components of effective response to climate change Clean water and sanitation; safe and adequate food; immunization; disease surveillance and response; safe and effective disease vector control; and disaster preparedness are all critical components of public health practices that are also adaptations to climate change. These programmes need to be strengthened globally with special concentration of effort in high-risk locations and populations in order to prevent climate-related injury, disease and death.

Cross-sector, interdisciplinary partnerships are necessary to meet this global health threat

Climate change is wide ranging, and effective adaptation will require the building of partnerships to leverage the expertise of government agencies, intergovernmental and nongovernmental organizations, industry and professional groups and local communities. Decisions affecting urban planning, transport, energy supply, food production, land use and water resources affect both climate and health. Collaboration across all these sectors is needed to find the innovative and effective solutions that will stabilize climate and protect health.

Action must begin now to protect health by applying both adaptation and mitigation

Scientific uncertainty persists about the possibility and timing of abrupt and catastrophic climate change if temperatures continue to rise. This makes it urgent for action to begin now to stabilize the climate through strong and effective mitigation undertaken simultaneously with adaptation activities to prevent increases in foreseeable climate-related illnesses. Full participation of the health sector in national and international processes for mitigation and adaptation to climate change is essential.

FACT SHEET

1. Over the last 50 years, human activities - particularly the burning of fossil fuels - have released sufficient quantities of carbon dioxide and other greenhouse gases to affect the global climate. The atmospheric concentration of carbon dioxide has increased by more than 30% since pre-industrial times, trapping more heat in the lower atmosphere. The resulting changes in the global climate bring a range of risks to health, from deaths in extreme high temperatures to changing patterns of infectious diseases.
2. From the tropics to the arctic, climate and weather have powerful direct and indirect impacts on human life. Weather extremes - such as heavy rains, floods, and disasters like Hurricane Katrina that devastated New Orleans, USA in August 2005 - endanger health as well as destroy property and livelihoods.

Approximately 600,000 deaths occurred worldwide as a result of weather-related natural disasters in the 1990s, some 95% of which took place in developing countries.

3. Intense short-term fluctuations in temperature can also seriously affect health - causing heat stress (hyperthermia) or extreme cold (hypothermia) - and lead to increased death rates from heart and respiratory diseases. Recent studies suggest that the record high temperatures in western Europe in the summer of 2003 were associated with a spike of an estimated 70,000 more deaths than the equivalent periods in previous years.

4. Increasing global temperatures affect levels and seasonal patterns of both man-made and natural air-borne particles, such as plant pollen, which can trigger asthma. About 300 million people suffer from asthma, and 255,000 people died of the disease in 2005. Asthma deaths are expected to increase by almost 20% in the next 10 years if urgent actions to curb climate change and prepare for its consequences are not taken.

5. Rising sea levels - another outcome of global warming - increase the risk of coastal flooding, and could cause population displacement. More than half of the world's population now lives within 60 kilometres of shorelines. Some of the most vulnerable regions are the Nile delta in Egypt, the Ganges-Brahmaputra delta in Bangladesh, and small island nations such as the Maldives in the Indian Ocean, and the Marshall Islands and Tuvalu in the Pacific Ocean. Floods can directly cause injury and death, and increase risks of infection from water and vector-borne diseases. Population displacement could increase tensions and potentially the risks of conflict.

6. More variable rainfall patterns are likely to compromise the supply of fresh water. Globally, water scarcity already affects four out of every 10 people. A lack of water and poor water quality can compromise hygiene and health. This increases the risk of diarrhoea, which kills approximately 1.8 million people every year, as well as trachoma (an eye infection that can lead to blindness) and other illnesses.

7. Water scarcity encourages people to transport water long distances and store supplies in their homes. This can increase the risk of household water contamination, causing illnesses.

8. Climatic conditions affect diseases transmitted through water, and via vectors such as mosquitoes. Climate-sensitive diseases are among the largest global killers. Diarrhoea, malaria and protein-energy malnutrition alone caused more than 3 million deaths globally in 2002, with over one third of these deaths occurring in Africa.

9. Malnutrition causes millions of deaths each year, from both a lack of sufficient nutrients to sustain life and a resulting vulnerability to infectious diseases such as malaria, diarrhoea, and respiratory illnesses. Increasing temperatures on the planet and more variable rainfalls are expected to reduce crop yields in many tropical developing regions, where food security is already a problem. Mali is a good example. Unless adaptive measures are taken, climate change is projected to approximately double by 2050 the percentage of its population at risk of hunger and associated health effects

10. Steps to reduce greenhouse gas emissions or lessen the health impacts of climate change could have positive health effects. For example, promoting the safe use of public transportation and active movement - such as biking or walking as alternatives to using private vehicles - could reduce carbon dioxide emissions and improve public health. They can not only cut traffic injuries, but also air pollution and associated respiratory and cardiovascular diseases. Increased levels of physical activity can lower overall mortality rates.

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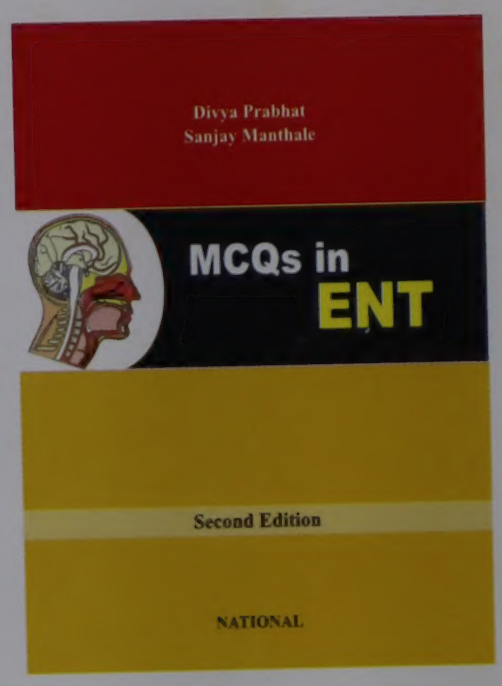

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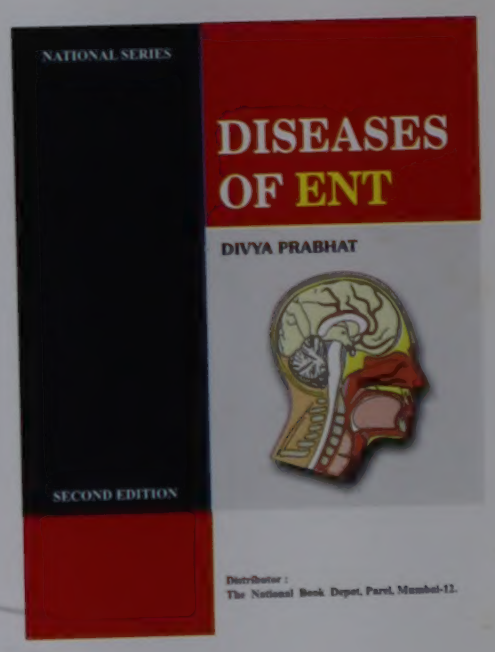

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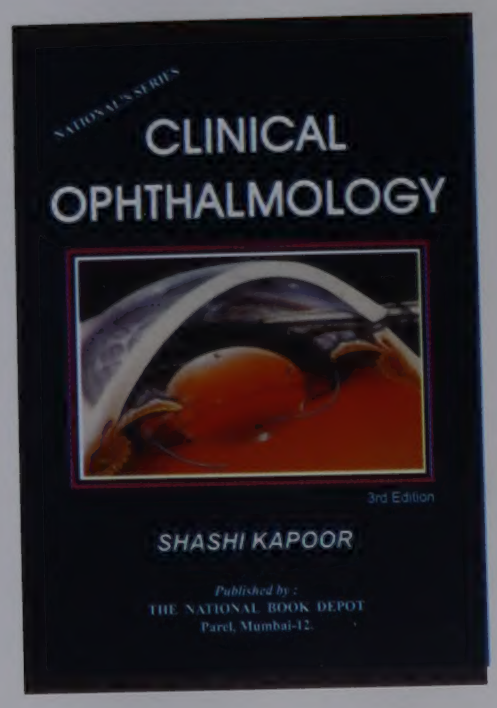

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